EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	1	"6723520"	USPAT	OR	OFF	2007/02/08 14:17
L2	1488	(wei.in. or gish.in. or schall.in. or vicari.in. or zlotnik.in.) and (cytokine or TECK)	US-PGPUB; USPAT	OR	ON	2007/02/08 14:18
L3	57	(wei.in. or gish.in. or schall.in. or vicari.in. or zlotnik.in.) and TECK	US-PGPUB; USPAT	OR	ON	2007/02/08 14:19
L4	44	(wang.in. or gish.in. or schall.in. or vicari.in. or zlotnik.in.) and TECK	US-PGPUB; USPAT	OR	ON	2007/02/08 14:35
L5	1389	teck or ccr9 or ccl25	US-PGPUB; USPAT	OR	ON	2007/02/08 14:36
L6	170527	antibod$	US-PGPUB; USPAT	OR	ON	2007/02/08 14:36
L7	90	5 same 6	US-PGPUB; USPAT	OR	ON	2007/02/08 14:36
L8	72802	gastrointest$ or crohn$	US-PGPUB; USPAT	OR	ON	2007/02/08 14:36
L9	4	7 same 8	US-PGPUB; USPAT	OR	ON	2007/02/08 14:36

Welcome to DIALOG

Dialog level 05.15.00D
? b 411;set files biotech
09feb07 09:02:17 User219511 Session D674.2
$0.00 0.117 DialUnits File410
$0.00 Estimated cost File410
$0.02 TELNET
$0.02 Estimated cost this search
$0.47 Estimated total session cost 0.245 DialUnits
File 411:DIALINDEX(R)

DIALINDEX(R)
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*** DIALINDEX search results display in an abbreviated ***
*** format unless you enter the SET DETAIL ON command. ***
You have 26 files in your file list.
(To see banners, use SHOW FILES command)
? s (teck or ccl25) and (gastrointest? or intest? or colon or bowel or crohn)

Your SELECT statement is:
s (teck or ccl25) and (gastrointest? or intest? or colon or bowel or crohn)

Items	File
68	5: Biosis Previews(R)_1969-2007/Feb W1
1	8: Ei Compendex(R)_1884-2007/Jan W4
25	24: CSA Life Sciences Abstracts_1966-2007/Nov
53	34: SciSearch(R) Cited Ref Sci_1990-2007/Feb W1
1	45: EMCare_2007/Feb W1
37	71: ELSEVIER BIOBASE_1994-2007/Feb W1
46	73: EMBASE_1974-2007/Feb 09
3	94: JICST-EPlus_1985-2007/Feb W3
2	98: General Sci Abs_1984-2007/Feb
6	135: NewsRx Weekly Reports_1995-2007/Feb W1
3	143: Biol. & Agric. Index_1983-2007/Jan
9	144: Pascal_1973-2007/Jan W4
44	155: MEDLINE(R)_1950-2007/Jan 26
2	266: FEDRIP_2006/Dec
7	357: Derwent Biotech Res.__1982-2007/Feb W1
40	399: CA SEARCH(R)_1967-2007/UD=14607

16 files have one or more items; file list includes 26 files.

? save temp; b 155,5,71,73,357,399;exs;rd
Temp SearchSave "TA374784765" stored
09feb07 09:03:24 User219511 Session D674.3
$2.99 1.016 DialUnits File411
$2.99 Estimated cost File411
$0.53 TELNET
$3.52 Estimated cost this search
$3.99 Estimated total session cost 1.261 DialUnits

SYSTEM:OS - DIALOG OneSearch
File 155:MEDLINE(R) 1950-2007/Jan 26
(c) format only 2006 Dialog
*File 155: MEDLINE has resumed updating with UD20061209. Please see HELP NEWS 154 for details.
File 5:Biosis Previews(R) 1969-2007/Feb W1
(c) 2007 The Thomson Corporation
*File 5: In preparation for coming enhancements, accession numbers will change soon. See HELP NEWS 5 for details.
File 71:ELSEVIER BIOBASE 1994-2007/Feb W1
(c) 2007 Elsevier B.V.
File 73:EMBASE 1974-2007/Feb 09
(c) 2007 Elsevier B.V.
File 357:Derwent Biotech Res. _1982-2007/Feb W1
(c) 2007 The Thomson Corp.
File 399:CA SEARCH(R) 1967-2007/UD=14607
(c) 2007 American Chemical Society
*File 399: Use is subject to the terms of your user/customer agreement.
IPCR/8 classification codes now searchable as IC=. See HELP NEWSIPCR.

Set	Items	Description
Executing TA374784765		
Hilight option is not available in file(s) 399		
HILIGHT set on as '%'		
	346	TECK
	348	CCL25
	549862	GASTROINTEST?
	1040434	INTEST?
	392275	COLON
	199451	BOWEL
	83826	CROHN
S1	242	(TECK OR CCL25) AND (GASTROINTEST? OR INTEST? OR COLON OR BOWEL OR CROHN)
S2	115	RD (unique items)

? t s2/7/1-115

2/7/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

22655372 PMID: 16804399
Chemokines involved in protection from colitis by CD4+CD25+ regulatory T cells.
Kristensen Nanna Ny; Brudzewsky Dan; Gad Monika; Claesson Mogens Helweg
Department of Medical Anatomy, University of Copenhagen, The Panum Institute, Copenhagen, Denmark. n.n.kristensen@mai.ku.dk
Inflammatory bowel diseases (United States) Jul 2006, 12 (7) p612-8, ISSN 1078-0998--Print Journal Code: 9508162
Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Chemokines are small proteins involved in the direction of migration of immune cells both during normal homeostasis and inflammation. Chemokines have been implicated in the pathology of many different inflammatory disorders and are therefore appealing therapeutic targets. Using a chemokine/chemokine receptor-specific gene expression profiling system of 67 genes, the authors have determined the expression profile of chemokine and chemokine receptor genes in the rectum of colitic mice and in mice that have been protected fromcolitis by CD4CD25 regulatory T cells. In mice protected from colitis, the authors found down regulation of the mRNA expression of the inflammatory chemokine receptors CCR1 and CXCR3 and their ligands CXCL9, CXCL10, CCL5, and CCL7. Also the transcripts for CCR9, %CCL25% , CCL17, and CXCL1 are found down regulated in protected compared with colitic animals. In addition, the authors' results suggest that CCL20 is used by CCR6 regulatory T cells in the complex process of controlling colitis because transcripts for this chemokine were expressed to a higher level in protected animals. The chemokine pathways identified in the present study may be of importance for the development of new targets for anti-inflammatory treatment strategies in human inflammatory %bowel% disease.
Record Date Created: 20060628
Record Date Completed: 20070116

2/7/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

22454494 PMID: 17101325
Antibody blockade of %CCL25%/CCR9 ameliorates early but not late chronic murine ileitis.
Rivera-Nieves Jesus; Ho Johnson; Bamias Giorgos; Ivashkina Natalia; Ley

Klaus; Oppermann Martin; Cominelli Fabio
Digestive Health Center of Excellence, Department of Internal Medicine, University of Virginia, Charlottesville, Virginia, USA. jr3u@virginia.edu
Gastroenterology (United States) Nov 2006, 131 (5) p1518-29, ISSN 0016-5085--Print Journal Code: 0374630
Contract/Grant No.: DK057880; DK; NIDDK; DK067254; DK; NIDDK
Publishing Model Print-Electronic
Document type: Journal Article; Research Support, N.I.H., Extramural; Research Support, Non-U.S. Gov't
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
BACKGROUND & AIMS: %CCL25% mediates the homeostatic recruitment of CCR9-expressing lymphocytes to the small %intestine%, but the function of this chemokine/receptor pair during chronic small %intestinal% inflammation has yet to be determined. Furthermore, although clinical trials to evaluate the efficacy of targeting the %CCL25%/CCR9 axis for the treatment of %Crohn%'s disease are being conducted, preclinical data in animal models of IBD are lacking. METHODS: In the current studies, we investigated the expression of %CCL25% and CCR9 as a function of disease progression in a spontaneous murine model of chronic ileitis (SAMP1/YitFc) using flow cytometry, real-time reverse-transcription polymerase chain reaction, enzyme-linked immunosorbent assay, and immunohistochemistry. In addition, we assessed the functional role of the axis in the overall disease process through therapeutic studies that target the chemokine or the receptor during early and late disease. RESULTS: The percentage of CCR9-expressing lymphocytes increased during early disease, accompanied by the appearance of a population of CCR9(high) lymphocytes, predominantly within CD8(+) T cells. Yet different from patients with primary sclerosing cholangitis, the expression of %CCL25% remained restricted to the small %intestine%, even in mice with inflammation of the biliary tree. Neutralization of the receptor or the chemokine attenuated early disease but showed no therapeutic efficacy during the later stages, when overall CCR9 expression decreased and the CCR9(high) population was absent. CONCLUSIONS: Our studies show that the role of this chemokine axis is not limited to homeostatic recruitment, as previously believed. However, these molecules appear to play their most crucial role during the early stages of chronic murine ileitis.
Record Date Created: 20061114
Record Date Completed: 20061212
Date of Electronic Publication: 20060816

2/7/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

21874161 PMID: 16839611
Expression of %TECK%/%CCL25% and MEC/CCL28 chemokines and their respective receptors CCR9 and CCR10 in porcine mucosal tissues.
Meurens Francois; Berri Mustapha; Whale Julia; Dybvig Tova; Strom Stacy; Thompson David; Brownlie Robert; Townsend Hugh G G; Salmon Henri; Gerdts Volker
Vaccine and Infectious Disease Organization, University of Saskatchewan, 120 Veterinary Road, Saskatoon, Saskatchewan, Canada S7N 5E3.
Veterinary immunology and immunopathology (Netherlands) Oct 15 2006, 113 (3-4) p313-27, ISSN 0165-2427--Print Journal Code: 8002006
Publishing Model Print-Electronic
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
%CCL25% and CCL28 (also named %TECK% and MEC) are CC chemokines primarily expressed by thymic dendritic cells and mucosal epithelial cells. The cognate receptors of %CCL25% and CCL28, named CCR9 and CCR10, are mainly expressed on T lymphocytes for CCR9 and IgA(+) and IgM(+) plasmablasts for CCR9 and CCR10, respectively. In human and mouse, chemokines %CCL25% and CCL28 play an important role in attracting immune cells to the %gastrointestinal% tract and in controlling segmental specialization of the %intestinal% immune system. To investigate if %CCL25% and CCL28 play a similar role in the pig and to better understand lymphocyte trafficking in this species, we cloned porcine %CCL25% and CCR10 and measured expression of %CCL25% , CCL28, CCR9, and CCR10 transcripts by real-time and conventional PCR in various tissues from newborn and young piglets, and adult sows. The results of the expression analyses show that (i) expression of %CCL25% mRNA is mainly restricted to the small %intestine%, (ii) CCL28 mRNA expression is detectable in all tested epithelial mucosal surfaces with the highest levels of expression in the mammary gland, trachea and large %intestine% , (iii) high levels of expression of CCR9 mRNA in CD3+ T lymphocytes, gut-associated lymphoid tissues (GALT), and the small %intestine% , (iv) high levels of expression of CCR10 mRNA in GALT, the large %intestine%, the small %intestine%, and the mammary gland, and (v) up-regulation of CCL28 mRNA expression during lactation in the mammary gland. This pattern of expression, which is discussed in the context of compartmentalization of the porcine common mucosal immune system into upper aero-digestive tract, small %intestine% and large %intestine%, suggests a key role for CCL28 in the recruitment of IgA secreting cells into the mammary gland enabling the passive transfer of IgA antibodies from mother to infant.
Record Date Created: 20060829
Record Date Completed: 20061024
Date of Electronic Publication: 20060712

2/7/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

20996939 PMID: 16670280
Redundant role of chemokines %CCL25%/%TECK% and CCL28/MEC in IgA+ plasmablast recruitment to the %intestinal% lamina propria after rotavirus infection.
Feng Ningguo; Jaimes Maria C; Lazarus Nicole H; Monak Denise; Zhang Caiqui; Butcher Eugene C; Greenberg Harry B
Department of Medicine, Stanford University School of Medicine, Stanford, CA 94305, USA.
Journal of immunology (Baltimore, Md. - 1950) (United States) May 15 2006, 176 (10) p5749-59, ISSN 0022-1767--Print Journal Code: 2985117R
Contract/Grant No.: AI 21362-20; AI; NIAID; AI 47822; AI; NIAID; DK 56339 ; DK; NIDDK
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Rotaviruses (RV) are the most important cause of severe childhood diarrheal disease. In suckling mice, infection with RV results in an increase in total and virus-specific IgA(+) plasmablasts in the small %intestinal% lamina propria (LP) soon after infection, providing a unique opportunity to study the mechanism of IgA(+) cell recruitment into the small %intestine% . In this study, we show that the increase in total and RV-specific IgA(+) plasmablasts in the LP after RV infection can be blocked by the combined administration of Abs against chemokines %CCL25% and CCL28, but not by the administration of either Ab alone. RV infection in CCR9 knockout mice still induced a significant accumulation of IgA(+) plasmablasts in the LP, which was blocked by the addition of anti-CCL28 Ab, confirming the synergistic role of %CCL25% and CCL28. The absence of IgA(+) plasmablast accumulation in LP following combined anti-chemokine treatment was not due to changes in proliferation or apoptosis in these cells. We also found that coadministration of anti-%CCL25% and anti-CCL28 Abs with the addition of anti-alpha(4) Ab did not further inhibit IgA(+) cell accumulation in the LP and that the %CCL25% receptor, CCR9, was coexpressed with the %intestinal% homing receptor alpha(4)beta(7) on IgA(+) plasmablasts. Finally, we showed that RV infection was associated with an increase in both %CCL25% and CCL28 in the small %intestine%. Hence, our findings indicate that alpha(4)beta(7) along with either CCR9 or CCR10 are sufficient for mediating the %intestinal% migration of IgA(+) plasmablasts during RV infection.
Record Date Created: 20060503
Record Date Completed: 20060622

2/7/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

20959176 PMID: 16582913
CD8+ recent thymic emigrants home to and efficiently repopulate the small %intestine% epithelium.
Staton Tracy L; Habtezion Aida; Winslow Monte M; Sato Tohru; Love Paul E; Butcher Eugene C
Program in Immunology, Department of Pathology, Stanford University School of Medicine, Stanford, California 94305, USA.
Nature immunology (United States) May 2006, 7 (5) p482-8, ISSN 1529-2908--Print Journal Code: 100941354
Contract/Grant No.: AI47822; AI; NIAID; DK060000; DK; NIDDK; DK07056; DK; NIDDK; DK56339; DK; NIDDK; GM37734; GM; NIGMS
Publishing Model Print-Electronic
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Prevailing knowledge dictates that naive alphabeta T cells require activation in lymphoid tissues before differentiating into effector or memory T cells capable of trafficking to nonlymphoid tissues. Here we demonstrate that CD8(+) recent thymic emigrants (RTEs) migrated directly into the small %intestine%. CCR9, %CCL25% and alpha(4)beta(7) integrin were required for gut entry of CD8(+) RTEs. After T cell receptor stimulation, %intestinal% CD8(+) RTEs proliferated and acquired a surface phenotype resembling that of intraepithelial lymphocytes. CD8(+) RTEs efficiently populated the gut of lymphotoxin-alpha-deficient mice, which lack lymphoid organs. These studies challenge the present understanding of naive alphabeta T cell trafficking and suggest that RTEs may be involved in maintaining a diverse immune repertoire at mucosal surfaces.
Record Date Created: 20060419
Record Date Completed: 20060616
Date of Electronic Publication: 20060402

2/7/6 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

20491671 PMID: 16517733
Functional characterization of the %CCL25% promoter in small %intestinal% epithelial cells suggests a regulatory role for caudal-related homeobox (Cdx) transcription factors.
Ericsson Anna; Kotarsky Knut; Svensson Marcus; Sigvardsson Mikael; Agace William
Immunology Section, Stem Cell Center, Biomedical Centre I-13, Lund University, S-22184 Lund, Sweden.
Journal of immunology (Baltimore, Md. - 1950) (United States) Mar 15 2006, 176 (6) p3642-51, ISSN 0022-1767--Print Journal Code: 2985117R
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The chemokine %CCL25% is selectively and constitutively expressed in the small %intestinal% epithelium and plays an important role in mediating lymphocyte recruitment to this site. In this study, we demonstrate that %CCL25% expression in murine small %intestinal% epithelial cells is independent of signaling through the lymphotoxin beta receptor and is not enhanced by inflammatory stimuli, pathways involved in driving the expression of most other chemokines. We define a transcriptional start site in the %CCL25% gene and a region -141 to -5 proximal of exon 1 that is required for minimal promoter activity in the small %intestinal% epithelial cell lines, MODE-K and mICc12. These cell lines expressed far less %CCL25% mRNA than freshly isolated small %intestinal% epithelial cells indicating that they are missing important factors driving %CCL25% expression. The %CCL25% promoter contained putative binding sites for the %intestinal% epithelial-associated Caudal-related homeobox (Cdx) transcription factors Cdx-1 and Cdx-2, and small %intestinal% epithelial cells but not MODE-K and mICc12 cells expressed Cdx-1 and Cdx-2. EMSA analysis demonstrated that Cdx proteins were present in nuclear extracts from freshly isolated small %intestinal% epithelial cells but not in MODE-K or mICcl2 cells, and bound to putative Cdx sites within the %CCL25% promoter. Finally, cotransfection of MODE-K cells with Cdx transcription factors significantly increased %CCL25% promoter activity as well as endogenous %CCL25% mRNA levels. Together these results demonstrate a unique pattern of regulation for %CCL25% and suggest a role for Cdx proteins in regulating %CCL25% transcription.
Record Date Created: 20060306
Record Date Completed: 20060424

2/7/7 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

20236654 PMID: 16498453
Aberrant homing of mucosal T cells and extra-%intestinal% manifestations of inflammatory %bowel% disease.
Adams David H; Eksteen Bertus
Liver Research Laboratories, MRC Centre for Immune Regulation, 5th Floor, Institute for Biomedical Research, Medical School, University of Birmingham, Edgbaston, Birmingham B15 2TT, UK. d.h.adams@bham.ac.uk
Nature reviews. Immunology (England) Mar 2006, 6 (3) p244-51, ISSN 1474-1733--Print Journal Code: 101124169;
Contract/Grant No.: Wellcome Trust
Publishing Model Print
Document type: Journal Article; Review
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Active inflammatory %bowel% disease (IBD) is often associated with simultaneous inflammation in the skin, eyes and joints. Inflammatory disease in the liver can also occur in patients with IBD but seems to be independent of inflammation in the %bowel%. In this Opinion article, we propose that the hepatic complications of IBD are mediated by long-lived mucosal T cells that are recruited to the liver in response to aberrantly expressed endothelial-cell adhesion molecules and chemokines that are normally restricted to the gut. Similar mechanisms might explain why certain diseases are associated with site-specific tissue distributions and might point to new therapeutic strategies that are based on modulating tissue-specific lymphocyte homing. (110 Refs.)
Record Date Created: 20060224
Record Date Completed: 20060330

2/7/8 (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15419023 PMID: 15833102
Transient expression of CC chemokine %TECK% in the ovary during ovulation: its potential role in ovulation.
Zhou Cindy; Wu Jean; Borillo Jason; Torres Lisa; McMahon John; Bao Yongde ; Lou Ya-Huan
Department of Diagnostic Sciences, Dental Branch, School of Medicine, University of Texas Health Science Center at Houston, Houston, T 77030, USA.
American journal of reproductive immunology (New York, N.Y. - 1989) (Denmark) May 2005, 53 (5) p238-48, ISSN 1046-7408--Print Journal Code: 8912860
Contract/Grant No.: R01 HD95993; HD; NICHD; T32 HD07324; HD; NICHD
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM

Record type: MEDLINE; Completed
PROBLEM: Chemokine thymus-expressed chemokine (%TECK%), which is expressed exclusively in the thymus and small %intestine%, plays a critical role in T-cell development. Our previous study revealed its expression in the ovary also. This study investigated its ovarian expression during ovulatory process. METHOD OF STUDY: Super-ovulation was induced in young female CD1 mice by equine chorionic gonadotropin (eCG) and human chorionic gonadotropic (hCG). Ovarian %TECK% expression during ovulation was determined by: (1) reverse transcriptase-polymerase chain reaction (RT-PCR) at mRNA level, (2) Western blot and immunohistology at the protein level, and (3) leukocyte infiltration assay at the bioactive level. RESULTS: A transient, high-level expression of %TECK% in murine ovaries at the mRNA level during hCG-induced ovulation was detected. Sequencing of directly cloned PCR product confirmed the ovarian expression of %TECK%. The peak expression of %TECK% was observed at 10-12 hr post-hCG injection; real-time PCR revealed an 800-fold increase during its expression peak over 0 hr. The expressed ovarian %TECK% protein was readily detectable by Western blot. Immunohistochemistry localized %TECK% expression to the ovarian interstitial tissue surrounding, or in the theca layer of the mature follicles undergoing ovulatory process. Expression of %TECK% receptor, the CC chemokine receptor (CCR9) was also detected in the ovulating ovaries. Using in vitro leukocyte infiltration assay, we first demonstrated that ovaries undergoing the ovulatory process were able to selectively chemoattract mononuclear cells. Importantly, neutralization of %TECK% by the antibody resulted in a 85% reduction in the chemotactic activities of the ovaries. CONCLUSION: This study suggested that ovarian expression of %TECK% is under a tight hormonal regulation, and expressed %TECK% may be responsible for recruitment of mononuclear cells into the ovary to participate in the ovulatory process.
Record Date Created: 20050418
Record Date Completed: 20050802

2/7/9 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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15244941 PMID: 15681774
%CCL25% enhances CD103-mediated lymphocyte adhesion to E-cadherin.
Ericsson Anna; Arya Anu; Agace William
Immunology Section, Department of Cell and Molecular Biology, Lund University, BMC I-13, SE-22184 Lund, Sweden. Anna.Ericsson@immuno.lu.se
Annals of the New York Academy of Sciences (United States) Dec 2004, 1029 p334-6, ISSN 0077-8923--Print Journal Code: 7506858
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Our results demonstrate that (1) CD103 is upregulated on CD8(+) T cells subsequent to their entry into the small %intestinal% epithelium, and (2) that the chemokine %CCL25% enhances CD103-mediated adhesion to E-cadherin. These results suggest a novel role for chemokines in modulating interactions between lymphocytes and epithelial cells at mucosal surfaces.
Record Date Created: 20050131
Record Date Completed: 20050421

2/7/10 (Item 10 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

15204358 PMID: 15557349
Hepatic endothelial %CCL25% mediates the recruitment of CCR9+ gut-homing lymphocytes to the liver in primary sclerosing cholangitis.
Eksteen Bertus; Grant Allister J; Miles Alice; Curbishley Stuart M; Lalor Patricia F; Hubscher Stefan G; Briskin Michael; Salmon Mike; Adams David H
Liver Research Laboratories, Institute for Biomedical Research, University of Birmingham, Queen Elizabeth Hospital, Birmingham B15 2TT, England, UK.
Journal of experimental medicine (United States) Dec 6 2004, 200 (11) p1511-7, ISSN 0022-1007--Print Journal Code: 2985109R
Publishing Model Print-Electronic
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Primary sclerosing cholangitis (PSC), a chronic inflammatory liver disease characterized by progressive bile duct destruction, develops as an extra-%intestinal% complication of inflammatory %bowel% disease (IBD) (Chapman, R.W. 1991. Gut. 32:1433-1435). However, the liver and %bowel% inflammation are rarely concomitant, and PSC can develop in patients whose colons have been removed previously. We hypothesized that PSC is mediated by long-lived memory T cells originally activated in the gut, but able to mediate extra-%intestinal% inflammation in the absence of active IBD (Grant, A.J., P.F. Lalor, M. Salmi, S. Jalkanen, and D.H. Adams. 2002. Lancet. 359:150-157). In support of this, we show that liver-infiltrating lymphocytes in PSC include mucosal T cells recruited to the liver by aberrant expression of the gut-specific chemokine %CCL25% that activates alpha4beta7 binding to mucosal addressin cell adhesion molecule 1 on the hepatic endothelium. This is the first demonstration in humans that T cells activated in the gut can be recruited to an extra-%intestinal% site of disease and provides a paradigm to explain the pathogenesis of extra-%intestinal% complications of IBD.
Record Date Created: 20041207
Record Date Completed: 20050112
Date of Electronic Publication: 20041122

2/7/11 (Item 11 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

15096474 PMID: 15452217
Maturation and trafficking markers on rotavirus-specific B cells during acute infection and convalescence in children.
Jaimes Maria C; Rojas Olga L; Kunkel Eric J; Lazarus Nicole H; Soler Dulce; Butcher Eugene C; Bass Dorsey; Angel Juana; Franco Manuel A; Greenberg Harry B
V.A. Palo Alto Health Care System, 3801 Miranda Ave., MC154C, Palo Alto, CA 94304, USA.
Journal of virology (United States) Oct 2004, 78 (20) p10967-76, ISSN 0022-538X--Print Journal Code: 0113724
Contract/Grant No.: AI 47822; AI; NIAID; DK 56339; DK; NIDDK; HL 67674; HL; NHLBI; R01 AI 21362-20; AI; NIAID
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
We have previously studied B cells, from people and mice, that express rotavirus-specific surface immunoglobulin (RV-sIg) by flow cytometry with recombinant virus-like particles that contain green fluorescent protein. In the present study we characterized circulating B cells with RV-sIg in children with acute and convalescent infection. During acute infection, circulating RV-sIgD(-) B cells are predominantly large, CD38(high), CD27(high), CD138(+/-), CCR6(-), alpha4beta7(+), CCR9(+), CCR10(+), cutaneous lymphocyte antigen-negative (CLA(-)), L-selectin(int/-), and sIgM(+), sIgG(-), sIgA(+/-) lymphocytes. This phenotype likely corresponds to gut-targeted plasma cells and plasmablasts. During convalescence the phenotype switches to small and large lymphocytes, CD38(int/-), CD27(int/-), CCR6(+), alpha4beta7(+/-), CCR9(+/-) and CCR10(-), most likely representing RV-specific memory B cells with both gut and systemic trafficking profiles. Of note, during acute RV infection both total and RV-specific murine IgM and IgA antibody-secreting cells migrate efficiently to CCL28 (the CCR10 ligand) and to a lesser extent to %CCL25% (the CCR9 ligand). Our results show that CCR10 and CCR9 can be expressed on IgM as well as IgA antibody-secreting cells in response to acute %intestinal% infection, likely helping target these cells to the gut. However, these %intestinal% infection-induced plasmablasts lack the CLA homing receptor

for skin, consistent with mechanisms of differential CCR10 participation in skin T versus %intestinal% plasma cell homing. Interestingly, RV memory cells generally lack CCR9 and CCR10 and instead express CCR6, which may enable recruitment to diverse epithelial sites of inflammation.
Record Date Created: 20040928
Record Date Completed: 20041102

2/7/12 (Item 12 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

15074037 PMID: 15368288
%CCL25%/CCR9 promotes the induction and function of CD103 on %intestinal% intraepithelial lymphocytes.
Ericsson Anna; Svensson Marcus; Arya Anu; Agace William W
Immunology Section, Department of Cell and Molecular Biology, Lund University, Lund, Sweden.
European journal of immunology (Germany) Oct 2004, 34 (10) p2720-9, ISSN 0014-2980--Print Journal Code: 1273201
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The integrin CD103 and the chemokine receptor CCR9 are co-expressed on small %intestinal% CD8(+) intraepithelial lymphocytes (IEL), naive murine CD8(+) T cells and by a small population of effector/memory CD8(+) T cells, indicating a potential role for CCR9 in regulating CD103 expression and function. Here, we demonstrate that CD103, in contrast to CCR9, is down-regulated on CD8(+) T cells following their activation in mesenteric lymph nodes and that effector CD8(+) T cells upon initial entry into the small %intestinal% epithelium are CCR9(+)CD103(-). CD103 was rapidly induced on wild-type CD8(+) T cells subsequent to their entry into the small %intestinal% epithelium, however, CCR9(-/-) CD8(+) T cells exhibited a significant delay in CD103 induction at this site. In addition, the CCR9 ligand, %CCL25%, that is constitutively expressed in the small %intestinal% epithelium, induced transient, dose-dependent and pertussis toxin-sensitive CD103-mediated adhesion of CD8(+) small %intestinal% IEL to a murine E-cadherin human Fc (mEFc) fusion protein. Together, these results demonstrate a role for CCR9/%CCL25% in promoting the induction and function of CD103 on CD8(+) IEL and suggest that this chemokine receptor/chemokine pair may function to regulate lymphocyte-epithelial interactions in the small %intestinal% mucosa.
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Record Date Completed: 20041119

2/7/13 (Item 13 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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15064877 PMID: 15356112
CC chemokine ligands 25 and 28 play essential roles in %intestinal% extravasation of IgA antibody-secreting cells.
Hieshima Kunio; Kawasaki Yuri; Hanamoto Hitoshi; Nakayama Takashi; Nagakubo Daisuke; Kanamaru Akihisa; Yoshie Osamu
Department of Microbiology, Kinki University School of Medicine, Osaka, Japan.
Journal of immunology (Baltimore, Md. - 1950) (United States) Sep 15 2004, 173 (6) p3668-75, ISSN 0022-1767--Print Journal Code: 2985117R
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
%CCL25% (also known as thymus-expressed chemokine) and CCL28 (also known as mucosae-associated epithelial chemokine) play important roles in mucosal immunity by recruiting IgA Ab-secreting cells (ASCs) into mucosal lamina propria. However, their exact roles in vivo still remain to be defined. In this study, we first demonstrated in mice that IgA ASCs in small %intestine% expressed CCR9, CCR10, and CXCR4 on the cell surface and migrated to their respective ligands %CCL25%, CCL28, and CXCL12 (also known as stromal cell-derived factor 1), whereas IgA ASCs in %colon% mainly expressed CCR10 and CXCR4 and migrated to CCL28 and CXCL12. Reciprocally, the epithelial cells of small %intestine% were immunologically positive for %CCL25% and CCL28, whereas those of %colon% were positive for CCL28 and CXCL12. Furthermore, the venular endothelial cells in small %intestine% were positive for %CCL25% and CCL28, whereas those in %colon% were positive for CCL28, suggesting their direct roles in extravasation of IgA ASCs. Consistently, in mice orally immunized with cholera toxin (CT), anti-%CCL25% suppressed homing of CT-specific IgA ASCs into small %intestine%, whereas anti-CCL28 suppressed homing of CT-specific IgA ASCs into both small %intestine% and %colon% . Reciprocally, CT-specific ASCs and IgA titers in the blood were increased in mice treated with anti-%CCL25% or anti-CCL28. Anti-CXCL12 had no such effects. Finally, both %CCL25% and CCL28 were capable of enhancing alpha4 integrin-dependent adhesion of IgA ASCs to mucosal addressin cell adhesion molecule-1 and VCAM-1. Collectively, %CCL25% and CCL28 play essential roles in %intestinal% homing of IgA ASCs primarily by mediating their extravasation into %intestinal% lamina propria. Copyright 2004 The American Association of Immunologists, Inc.
Record Date Created: 20040909
Record Date Completed: 20041019

2/7/14 (Item 14 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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15026375 PMID: 15306587
CCR2 expressing CD4+ T lymphocytes are preferentially recruited to the ileum in %Crohn%'s disease.
Connor S J; Paraskevopoulos N; Newman R; Cuan N; Hampartzoumian T; Lloyd A R; Grimm M C
Department of Medicine, St George Clinical School, Kogarah, NSW 2217, Australia.
Gut (England) Sep 2004, 53 (9) p1287-94, ISSN 0017-5749--Print Journal Code: 2985108R
Publishing Model Print; Erratum in Gut. 2004 Nov;53(11) 1722
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
BACKGROUND AND AIMS: Chemokine receptors are key determinants of leucocyte trafficking. While the chemokine receptor CCR9 and its chemokine ligand %CCL25% (%TECK%) mediate lymphocyte homing to the healthy small %intestine%, the chemokine receptors important for recruitment during %intestinal% inflammation are undefined. Animal studies have suggested potential roles for CCR2 and CCR5 in inflammatory %bowel% disease (IBD). The aim of this study was to understand the role of CCR2 in human IBD. METHODS: Resections of ileum or %colon% were obtained from patients undergoing surgery for small %bowel% %Crohn%'s disease (SBCD; n = 10), %Crohn% 's colitis (n = 5), ulcerative colitis (n = 6), and non-IBD related conditions (control ileum n = 11; control %colon% n = 11). Expression of CCR2 by lamina propria lymphocytes (LPLs) was determined by both flow cytometry and immunohistochemistry. As a functional correlate, chemotaxis assays using the CCR2 ligand, CCL2 (MCP-1), were performed. Expression of CCR2 by peripheral blood lymphocytes was determined by flow cytometry. RESULTS: There were greater than 30-fold more CCR2(+) LPLs in SBCD than in control ileum (29.3% (19.9-55.1) v 0.9% (0.4-11.5); p = 0.0007). Specifically, CCR2(+)CD4(+) LPLs were increased (p = 0.002) whereas CCR2(+)CD8(+) LPLs were not. Increased expression included both memory (CD45RO(+); p = 0.005) and naive (CD45RO(-); p = 0.01) CCR2(+) populations. The increase in CCR2(+) LPLs in SBCD was confirmed by both immunohistochemistry (p = 0.0002) and enhanced chemotactic responses to CCL2. CCR2 expression was not increased in the peripheral blood of patients with SBCD, suggesting ongoing recruitment of the CCR2(+) population to the ileum. In contrast with SBCD, there was no significant increase in CCR2(+) LPLs in %Crohn%'s colitis or ulcerative colitis samples. CONCLUSIONS: The

chemokine receptor CCR2 appears to be an important contributor to accumulation of CD4(+) T lymphocytes in the ileum in small %bowel% %Crohn% 's disease. Blockade of CCR2 may provide a novel therapeutic alternative.
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Record Date Completed: 20041004

2/7/15 (Item 15 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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14883916 PMID: 15139741
Lymphocyte homing in the pathogenesis of extra-%intestinal% manifestations of inflammatory %bowel% disease.
Eksteen Bertus; Miles Alice E; Grant Allister J; Adams David H
Liver Research Laboratories, MRC Centre for Immune Regulation, University of Birmingham, Queen Elizabeth Hospital, Birmingham.
Clinical medicine (London, England) (England) Mar-Apr 2004, 4 (2) p173-80, ISSN 1470-2118--Print Journal Code: 101092853
Publishing Model Print
Document type: Journal Article; Review
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Inflammatory %bowel% disease is associated with extra-%intestinal% manifestations which occur either at the same time as flares of %bowel% inflammation (skin and eye disease) or run a course that is independent to inflammation in the %bowel% (liver and some joint syndromes). It has been suggested that the skin and eye complications occur as a consequence of the recruitment of activated effector cells released from the gut into the circulation to extra-%intestinal% site where they cause acute damage. However, this does not explain how patients can develop primary sclerosing cholangitis many years after having their %colon% removed for colitis. We propose that long-lived populations of memory lymphocytes arise as a consequence of %bowel% inflammation and that these cells express homing receptors that direct their subsequent migration not only to the gut but also to the liver. These long-lived cells may recirculate to the liver for many years and, in the absence of a local activating stimulus, will not cause damage. However, if they are subsequently activated in the liver this will lead to the development of inflammation and tissue damage which promotes the recruitment of more mucosal lymphocytes resulting in persistent inflammation and disease. The recent findings that MAdCAM-1 and %CCL25% , previously thought to be restricted to the gut, are up-regulated in the liver during inflammatory liver diseases that complicate IBD support the concept that common mechanisms control lymphocyte recruitment to the inflamed liver and gut. (20 Refs.)
Record Date Created: 20040513
Record Date Completed: 20040708

2/7/16 (Item 16 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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14841386 PMID: 15086554
Functional CCR9 expression is associated with small %intestinal% metastasis.
Letsch Anne; Keilholz Ulrich; Schadendorf Dirk; Assfalg Geraldine; Asemissen Anne Marie; Thiel Eckhard; Scheibenbogen Carmen
Charite University Medicine Berlin, Campus Benjamin-Franklin, Hematology, Oncology and Transfusion Medicine, Berlin, Germany. anne.letsch@medizin.fu-berlin.de
Journal of investigative dermatology (United States) Mar 2004, 122 (3) p685-90, ISSN 0022-202X--Print Journal Code: 0426720
Publishing Model Print; Comment in J Invest Dermatol. 2004 Mar;122(3) xiv-xv; Comment in PMID 15086589
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
In general, metastases to the small %intestine% are rare, and mostly occur in melanoma. CCR9 has been shown to be the principal chemokine receptor for the thymus expressed chemokine (%TECK%), a chemokine selectively expressed in the small %intestine% and thymus. Here we show that CCR9 is highly expressed on melanoma cells and all melanoma cell lines isolated from small %intestinal% metastases, and on a proportion of cell lines from other sites. Only melanoma cells and cell lines from small %intestinal% metastases, however, were responsive to the CCR9 ligand %TECK% , as assessed by receptor downregulation and by actin polymerization. CCR9 expression was also found on the adenocarcinoma cell line CaCo-2 expressing characteristics of enterocytic differentiation, but not on any other cell line isolated from colorectal, breast, and lung cancer. Our data provide evidence that the aberrant functional cell surface expression of an organ-specific chemokine receptor is associated with metastasis to this site. The regulation of receptor function seems to be a critical step in the metastatic process.
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Record Date Completed: 20040525

2/7/17 (Item 17 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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14740418 PMID: 14592943
Demonstration of functional role of %TECK%/%CCL25% in T lymphocyte-endothelium interaction in inflamed and uninflamed %intestinal% mucosa.
Hosoe Naoki; Miura Soichiro; Watanabe Chikako; Tsuzuki Yoshikazu; Hokari Ryota; Oyama Tokushige; Fujiyama Yoichi; Nagata Hiroshi; Ishii Hiromasa
Department of Internal Medicine, School of Medicine, Keio University, 35 Shinanomachi, Shinjukuku, Tokyo 160-8582, Japan.
American journal of physiology. Gastrointestinal and liver physiology (United States) Mar 2004, 286 (3) pG458-66, ISSN 0193-1857--Print Journal Code: 100901227
Publishing Model Print-Electronic
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
It has recently been suggested that C-C chemokines may play a role in the organ-specific homing of lymphocytes, but there is not enough in vivo evidence in %intestinal% mucosa. The aim of this study was to examine whether thymus-expressed chemokine (%TECK%)/%CCL25% and its ligand CCR9 are involved in T-lymphocyte interaction with microvessels of murine %intestinal% mucosa. T lymphocytes from the small %intestine% were fluorescence labeled, and their adhesion to mucosal microvessels was observed by intravital microscopy. Lamina proprial lymphocytes (LPL) and intraepithelial lymphocytes (IEL) adhered to both the small %intestine% and %colon%, and desensitization of CCR9 with %TECK%/%CCL25% or anti-%TECK%/ %CCL25% antibody significantly inhibited these adhesions only in small %intestine% . At both sites, TNF-alpha significantly increased LPL adhesion but not IEL adhesion. Desensitization of CCR9 or anti-%TECK%/%CCL25% antibody also attenuated the TNF-alpha-induced LPL adhesion in the small %intestine%. Increased expression of %TECK%/%CCL25% by TNF-alpha was observed in the lamina propria of small %intestine%. %TECK%/%CCL25% may thus play an important role in the adherence of mucosal lymphocytes to the microvessels of the small %intestine% but not the %colon% under uninflamed as well as inflamed conditions.
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Record Date Completed: 20040316
Date of Electronic Publication: 20031030

2/7/18 (Item 18 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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14734959 PMID: 14744993
Chemokine receptor CCR9 contributes to the localization of plasma cells

to the small %intestine%.
Pabst Oliver; Ohl Lars; Wendland Meike; Wurbel Marc-Andre; Kremmer Elisabeth; Malissen Bernard; Forster Reinhold
Institute of Immunology, Hannover Medical School, 30625 Hannover, Germany.
Journal of experimental medicine (United States) Feb 2 2004, 199 (3) p411-6, ISSN 0022-1007--Print Journal Code: 2985109R
Publishing Model Print-Electronic
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Humoral immunity in the gut-associated lymphoid tissue is characterized by the production of immunoglobulin A (IgA) by antibody-secreting plasma cells (PCs) in the lamina propria. The chemokine %CCL25% is expressed by %intestinal% epithelial cells and is capable of inducing chemotaxis of IgA+ PCs in vitro. Using a newly generated monoclonal antibody against murine CCR9, we show that IgA+ PCs express high levels of CCR9 in the mesenteric lymph node (MLN) and Peyer's patches (PPs), but down-regulate CCR9 once they are located in the small %intestine%. In CCR9-deficient mice, IgA+ PCs are substantially reduced in number in the lamina propria of the small %intestine%. In adoptive transfer experiments, CCR9-deficient IgA+ PCs show reduced migration into the small %intestine% compared with wild-type controls. Furthermore, CCR9 mutants fail to mount a regular IgA response to an orally administered antigen, although the architecture and cell type composition of PPs and MLN are unaffected and are functional for the generation of IgA PCs. These findings provide profound in vivo evidence that %CCL25%/CCR9 guides PCs into the small %intestine%.
Record Date Created: 20040203
Record Date Completed: 20040907
Date of Electronic Publication: 20040126

2/7/19 (Item 19 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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14674635 PMID: 15030305
Features and functions of gamma delta T lymphocytes: focus on chemokines and their receptors.
Kabelitz Dieter; Wesch Daniela
Institute of Immunology, UK S-H Campus Kiel, Michaelisstr. 5, D-24105 Kiel, Germany. kabelitz@immunologie.uni-kiel.de
Critical reviews in immunology (United States) 2003, 23 (5-6) p339-70, ISSN 1040-8401--Print Journal Code: 8914819
Publishing Model Print
Document type: Journal Article; Review
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
gamma delta T cells are a distinct subgroup of T lymphocytes that are enriched at certain anatomical localizations, such as the small %intestinal% epithelia and other epithelia. gamma delta T cells recognize microbial antigens, such as heat shock proteins (in mice) or phosphorylated bacterial metabolites (in humans), and control the integrity of epithelia. At the effector cell level, they share with the conventional alpha beta T lymphocytes potent cytotoxic activity and the capacity to produce a variety of cytokines, including specific cytokines such as keratinocyte growth factor. Here we summarize the current knowledge on the role of chemokines and their receptors in the migration and function of gamma delta T cells. As an example, the migration of gamma delta T cells to the small %intestine% is guided by the chemokine receptor CCR9 and the local expression of the corresponding ligand %CCL25% (also termed thymus-expressed chemokine, %TECK%). Chemokine receptor expression also correlates with the functional program of T cells. In this respect, the strong expression of the MIP-1 alpha/MIP-1 beta/RANTES (CCL3/CCL4/CCL5)-receptor CCR5 correlates with a T-helper 1 phenotype of human V gamma 9V delta 2-expressing gamma delta T cells. The regulation of chemokine receptors, together with the pattern of local chemokine production, plays an important role in the localization of gamma delta T cells under physiological and pathophysiological conditions, such as infection, inflammation, and tumor defense. (259 Refs.)
Record Date Created: 20040319
Record Date Completed: 20041005

2/7/20 (Item 20 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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14389626 PMID: 12851648
Chemokines in lymphocyte trafficking and %intestinal% immunity.
Kunkel Eric J; Campbell Daniel J; Butcher Eugene C
Laboratory of Immunology and Vascular Biology, Department of Pathology, Stanford University School of Medicine, Stanford, CA, USA. ekunkel@bioseekinc.com
Microcirculation (New York, N.Y. - 1994) (United States) Jun 2003, 10 (3-4) p313-23, ISSN 1073-9688--Print Journal Code: 9434935
Contract/Grant No.: AI-47822; AI; NIAID; DK-56339; DK; NIDDK; GM-37734; GM; NIGMS; HL-67674; HL; NHLBI
Publishing Model Print
Document type: Journal Article; Review
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Lymphocyte migration through gut-associated lymphoid tissues (GALT) and into %intestinal% effector sites is critical to %intestinal% immune system function and homeostasis. Chemokines contribute to lymphocyte trafficking by triggering integrin activation and firm arrest in the vasculature and mediating chemotactic localization within tissues. Several chemokines have been identified that are expressed in the GALT and/or the %intestines% themselves (%TECK%/%CCL25% , MEC/CCL28, and MIP-3alpha/CCL20) and play a role in %intestinal% lymphocyte localization, including unification of %intestinal% and other mucosa-associated effector sites; segmental specialization of the %intestines%; and subset selective localization to the %intestines% . This review examines the role of these chemokines (and their receptors CCR9, CCR10, and CCR6, respectively) in lymphocyte homing to the GALT, in the induction and differentiation of %intestinal% effector and memory lymphocytes, and in the homeostatic and inflammatory localization of lymphocytes to the %intestines%. (75 Refs.)
Record Date Created: 20030709
Record Date Completed: 20031106

2/7/21 (Item 21 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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14381059 PMID: 12840763
Selective imprinting of gut-homing T cells by Peyer's patch dendritic cells.
Mora J Rodrigo; Bono Maria Rosa; Manjunath N; Weninger Wolfgang; Cavanagh Lois L; Rosemblatt Mario; Von Andrian Ulrich H
The Center for Blood Research and Department of Pathology, Harvard Medical School, Boston, Massachusetts 02115, USA.
Nature (England) Jul 3 2003, 424 (6944) p88-93, ISSN 1476-4687--Electronic Journal Code: 0410462
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Whereas naive T cells migrate only to secondary lymphoid organs, activation by antigen confers to T cells the ability to home to non-lymphoid sites. Activated effector/memory T cells migrate preferentially to tissues that are connected to the secondary lymphoid organs where antigen was first encountered. Thus, oral antigens induce effector/memory cells that express essential receptors for %intestinal% homing, namely the integrin alpha4beta7 and CCR9, the receptor for the gut-associated chemokine %TECK%/%CCL25% (refs 6, 8, 9). Here we show that

this imprinting of gut tropism is mediated by dendritic cells from Peyer's patches. Stimulation of CD8-expressing T cells by dendritic cells from Peyer's patches, peripheral lymph nodes and spleen induced equivalent activation markers and effector activity in T cells, but only Peyer's patch dendritic cells induced high levels of alpha4beta7, responsiveness to %TECK% and the ability to home to the small %intestine%. These findings establish that Peyer's patch dendritic cells imprint gut-homing specificity on T cells, and thus license effector/memory cells to access anatomical sites most likely to contain their cognate antigen.
Record Date Created: 20030703
Record Date Completed: 20030801

2/7/22 (Item 22 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14360363 PMID: 12816994
CC chemokine receptor 9 expression defines a subset of peripheral blood lymphocytes with mucosal T cell phenotype and Th1 or T-regulatory 1 cytokine profile.
Papadakis Konstantinos A; Landers Carol; Prehn John; Kouroumalis Elias A; Moreno Sofia T; Gutierrez-Ramos Jose-Carlos; Hodge Martin R; Targan Stephan R
Burns and Allen Research Institute and Inflammatory Bowel Disease Center, Cedars-Sinai Medical Center, David Geffen School of Medicine at UCLA, Los Angeles, CA 90048, USA. Papadakis@cshs.org
Journal of immunology (Baltimore, Md. - 1950) (United States) Jul 1 2003, 171 (1) p159-65, ISSN 0022-1767--Print Journal Code: 2985117R
Contract/Grant No.: DK-46763; DK; NIDDK; DK56328; DK; NIDDK
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The chemokine receptor CCR9 is expressed on most small %intestinal% lamina propria and intraepithelial lymphocytes and on a small subset of peripheral blood lymphocytes. CCR9-expressing lymphocytes may play an important role in small %bowel% immunity and inflammation. We studied the phenotype and functional characteristics of CCR9(+) lymphocytes in blood from normal donors. A subset of CCR9(+) T cells have a phenotype of activated cells and constitutively express the costimulatory molecules CD40L and OX-40. In contrast to CCR9(-), CCR9(+)CD4(+) peripheral blood T cells proliferate to anti-CD3 or anti-CD2 stimulation and produce high levels of IFN-gamma and IL-10. IL-10-producing cells were exclusively detected within the CCR9(+) subset of CD4(+) T cells by intracellular staining and were distinct from IL-2- and IFN-gamma-producing cells. Moreover, memory CCR9(+)CD4(+) lymphocytes respond to CD2 stimulation with proliferation and IFN-gamma/IL-10 production, whereas memory CCR9(-)CD4(+) cells were unresponsive. In addition, memory CCR9(+)CD4(+) T cells support Ig production by cocultured CD19(+) B cells in the absence of prior T cell activation or addition of exogenous cytokines. Our data show that the memory subset of circulating CCR9(+)CD4(+) T cells has characteristics of mucosal T lymphocytes and contains cells with either Th1 or T-regulatory 1 cytokine profiles. Studies on the cytokine profile and Ag specificity of this cell subset could provide important insight into small %intestinal% immune-mediated diseases and oral tolerance in humans.
Record Date Created: 20030620
Record Date Completed: 20030922

2/7/23 (Item 23 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14270286 PMID: 12705880
Antigen-induced chemokine activation in mouse buccal epithelium.
Otten Kevin; Dragoo Jolene; Wang Heuy-Ching; Klein John R
Department of Basic Sciences, University of Texas Health Science Center, Dental Branch, Rm 4.133, 6516 M.D. Anderson Blvd., Houston, TX 77030, USA.
Biochemical and biophysical research communications (United States) Apr 25 2003, 304 (1) p36-40, ISSN 0006-291X--Print Journal Code: 0372516
Contract/Grant No.: DK35566; DK; NIDDK
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The oral mucosa is an active though poorly understood immunological site. Using an experimental animal system involving antigen priming into the oral mucosa of transgenic mice expressing T cell receptor (TCR) for a peptide antigen of hen-egg lysozyme (HEL), the expression of six chemokine receptors and seven chemokine ligands were studied before and after antigen exposure. Within 24h of local antigen priming, the expression of three chemokine receptor genes (CCR3; CCR5, and CCR7) and three chemokine ligand genes (CCL12, CCL19, and %CCL25%) were significantly upregulated. These included chemokines known to be responsible for the trafficking of T cells and other leukocytes into tissue sites. Additionally, expression of the chemokine ligand gene, %CCL25% (thymus-expressed chemokine [%TECK%]), which has been linked to T cell migration and/or local T cell development in the %intestine% , was also markedly elevated in buccal epithelia after antigen exposure. These findings define a process of selective activation of proinflammatory chemokines and/or their receptors following local antigen exposure, and they provide the first evidence, indicating that this may be accompanied by in situ development of T cells in oral tissues.
Record Date Created: 20030422
Record Date Completed: 20030520

2/7/24 (Item 24 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14242043 PMID: 12671049
CCR10 expression is a common feature of circulating and mucosal epithelial tissue IgA Ab-secreting cells.
Kunkel Eric J; Kim Chang H; Lazarus Nicole H; Vierra Mark A; Soler Dulce; Bowman Edward P; Butcher Eugene C
Laboratory of Immunology and Vascular Biology, Department of Pathology, Stanford University School of Medicine, Stanford, California, USA. ekunkel@bioseekinc.com
Journal of clinical investigation (United States) Apr 2003, 111 (7) p1001-10, ISSN 0021-9738--Print Journal Code: 7802877
Contract/Grant No.: AI-47822; AI; NIAID; DK-56339; DK; NIDDK; GM-37734; GM; NIGMS; HL-67674; HL; NHLBI
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The dissemination of IgA-dependent immunity between mucosal sites has important implications for mucosal immunoprotection and vaccine development. Epithelial cells in diverse %gastrointestinal% and nonintestinal mucosal tissues express the chemokine MEC/CCL28. Here we demonstrate that CCR10, a receptor for MEC, is selectively expressed by IgA Ab-secreting cells (large s/cIgA(+)CD38(hi)CD19(int/-)CD20(-)), including circulating IgA(+) plasmablasts and almost all IgA(+) plasma cells in the salivary gland, small %intestine%, large %intestine% , appendix, and tonsils. Few T cells in any mucosal tissue examined express CCR10. Moreover, tonsil IgA plasmablasts migrate to MEC, consistent with the selectivity of CCR10 expression. In contrast, CCR9, whose ligand %TECK%/ %CCL25% is predominantly restricted to the small %intestine% and thymus, is expressed by a fraction of IgA Ab-secreting cells and almost all T cells in the small %intestine%, but by only a small percentage of plasma cells and plasmablasts in other sites. These results point to a unifying role for CCR10 and its mucosal epithelial ligand MEC in the migration of circulating IgA plasmablasts and, together with other tissue-specific homing mechanisms, provides a mechanistic basis for the specific dissemination of IgA Ab-secreting cells after local immunization.
Record Date Created: 20030402

Record Date Completed: 20030505

2/7/25 (Item 25 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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14222959 PMID: 12646646
A common mucosal chemokine (mucosae-associated epithelial chemokine/CCL28) selectively attracts IgA plasmablasts.
Lazarus Nicole H; Kunkel Eric J; Johnston Brent; Wilson Eric; Youngman Kenneth R; Butcher Eugene C
Laboratory of Immunology and Vascular Biology, Department of Pathology, Stanford University School of Medicine, Stanford, CA 94305-5324, USA.
Journal of immunology (Baltimore, Md. - 1950) (United States) Apr 1 2003, 170 (7) p3799-805, ISSN 0022-1767--Print Journal Code: 2985117R
Contract/Grant No.: 5 T32 AI07290; AI; NIAID; AI-37832; AI; NIAID; AI-47822; AI; NIAID; DK10022; DK; NIDDK; DK56339; DK; NIDDK; GM-37734; GM; NIGMS; HL-57492; HL; NHLBI; HL-67674; HL; NHLBI
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
IgA immunoblasts can seed both %intestinal% and nonintestinal mucosal sites following localized mucosal immunization, an observation that has led to the concept of a common mucosal immune system. In this study, we demonstrate that the mucosae-associated epithelial chemokine, MEC (CCL28), which is expressed by epithelia in diverse mucosal tissues, is selectively chemotactic for IgA Ab-secreting cells (ASC): MEC attracts IgA- but not IgG- or IgM-producing ASC from both %intestinal% and nonintestinal lymphoid and effector tissues, including the %intestines%, lungs, and lymph nodes draining the bronchopulmonary tree and oral cavity. In contrast, the small %intestinal% chemokine, %TECK% (%CCL25%), attracts an overlapping subpopulation of IgA ASC concentrated in the small %intestines% and its draining lymphoid tissues. Surprisingly, T cells from mucosal sites fail to respond to MEC. These findings suggest a broad and unifying role for MEC in the physiology of the mucosal IgA immune system.
Record Date Created: 20030320
Record Date Completed: 20030708

2/7/26 (Item 26 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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14021183 PMID: 12442331
Involvement of %CCL25% (%TECK%) in the generation of the murine small-%intestinal% CD8alpha alpha+CD3+ intraepithelial lymphocyte compartment.
Marsal Jan; Svensson Marcus; Ericsson Anna; Iranpour Amir H; Carramolino Laura; Marquez Gabriel; Agace William W
Immunology Section, Department of Cell and Molecular Biology, Lund University, Lund, Sweden.
European journal of immunology (Germany) Dec 2002, 32 (12) p3488-97, ISSN 0014-2980--Print Journal Code: 1273201
Contract/Grant No.: DK-52978; DK; NIDDK
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The CC chemokine %CCL25% (%TECK%) is selectively expressed in the thymus and small %intestine% , indicating a potential role in T lymphocyte development. In the present study we examined the role of %CCL25% in the generation of the small-%intestinal% CD8alpha alpha(+)CD3(+) intraepithelial lymphocyte (IEL) compartment. %CCL25% mRNA expression in the murine small %intestine% increased at three weeks of age and corresponded with the appearance of CD8alpha alpha(+)CD3(+) lymphocytes in the small-%intestinal% epithelium. Administration of monoclonal neutralizing anti-%CCL25% antibody to two-week-old mice led to a approximately 50% reduction in the total number of CD8alpha alpha(+)TCRgamma delta(+) and CD8alpha alpha(+)TCRalpha beta(+) IEL at four weeks of age. Freshly isolated murine CD8alpha alpha(+)CD3(+) IEL migrated in response to %CCL25% and expressed the %CCL25% receptor, CCR9. Analysis of CCR9 expression on putative IEL precursor populations demonstrated the presence of both CCR9(-) and CCR9(+) cells and indicated that up-regulation of this receptor occurred during IEL precursor differentiation. Finally, data from wild-type and RAG(-/-) mice suggested that the reduction in CD8alpha alpha(+)CD3(+) IEL in anti-%CCL25% antibody treated mice resulted primarily from defective maintenance and/or development of IEL precursors rather than a direct effect on mature CD8alpha alpha(+)CD3(+) IEL.
Record Date Created: 20021120
Record Date Completed: 20030130

2/7/27 (Item 27 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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13978876 PMID: 12393847
%CCL25% mediates the localization of recently activated CD8alphabeta(+) lymphocytes to the small-%intestinal% mucosa.
Svensson Marcus; Marsal Jan; Ericsson Anna; Carramolino Laura; Broden Therese; Marquez Gabriel; Agace William W
Immunology Section, Department of Cell and Molecular Biology, Lund University, Lund, Sweden.
Journal of clinical investigation (United States) Oct 2002, 110 (8) p1113-21, ISSN 0021-9738--Print Journal Code: 7802877
Publishing Model Print; Comment in J Clin Invest. 2002 Oct;110(8) 1079-81 ; Comment in PMID 12393843
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The recruitment of antigen-specific T lymphocytes to the %intestinal% mucosa is central to the development of an effective mucosal immune response, yet the mechanism by which this process occurs remains to be fully defined. Here we show that the CC chemokine receptor 9 (CCR9) is selectively and functionally expressed on murine alpha(E)beta(7)(+) naive CD8alphabeta(+) lymphocytes and a subset of recently activated CD69(+) CD8alphabeta(+) lymphocytes. Using a T cell receptor transgenic transfer model, we demonstrate that CCR9 expression is functionally maintained on CD8alphabeta(+) lymphocytes following activation in mesenteric lymph nodes but rapidly downregulated on CD8alphabeta(+) lymphocytes activated in peripheral lymph nodes. These recently activated CCR9(+) CD8alphabeta(+) lymphocytes selectively localized to the small-%intestinal% mucosa, and in vivo neutralization of the CCR9 ligand, %CCL25%, reduced the ability of these cells to populate the small-%intestinal% epithelium. Together these results demonstrate an important role for chemokines in the localization of T lymphocytes to the small-%intestinal% mucosa and suggest that targeting %CCL25% and/or CCR9 may provide a means to selectively modulate small-%intestinal% immune responses.
Record Date Created: 20021023
Record Date Completed: 20021203

2/7/28 (Item 28 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

13978872 PMID: 12393843
%Intestinal% attraction: %CCL25% functions in effector lymphocyte recruitment to the small %intestine%.
Campbell Daniel J; Butcher Eugene C
Laboratory of Immunology and Vascular Biology, Department of Pathology, Stanford University School of Medicine, Stanford, California 94304, USA. daniel@macampbell.com
Journal of clinical investigation (United States) Oct 2002, 110 (8) p1079-81, ISSN 0021-9738--Print Journal Code: 7802877
Publishing Model Print; Comment on J Clin Invest. 2002 Oct;110(8) 1113-21

; Comment on PMID 12393847
Document type: Comment; Journal Article; Review
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
(20 Refs.)
Record Date Created: 20021023
Record Date Completed: 20021203

2/7/29 (Item 29 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

13834008 PMID: 12117869
Where do IgA plasma cells in the gut come from?
Golby S J C; Spencer J
Histopathology Department, Guy's, King's and St Thomas' School of Medicine, St Thomas' Campus, London SE1 7EH, UK.
Gut (England) Aug 2002, 51 (2) p150-1, ISSN 0017-5749--Print Journal Code: 2985108R
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Record Date Created: 20020715
Record Date Completed: 20020910

2/7/30 (Item 30 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

13816936 PMID: 12096027
Pivotal role of %CCL25% (%TECK%)-CCR9 in the formation of gut cryptopatches and consequent appearance of %intestinal% intraepithelial T lymphocytes.
Onai Nobuyuki; Kitabatake Masahiro; Zhang Yan-yun; Ishikawa Hiromichi; Ishikawa Sho; Matsushima Kouji
Department of Molecular Preventive Medicine and Core Research for Evolutional Science and Technology, Graduate School of Medicine, University of Tokyo, 7-3-1 Hongo, Bunkyo-ku, Tokyo 113-033, Japan.
International immunology (England) Jul 2002, 14 (7) p687-94, ISSN 0953-8178--Print Journal Code: 8916182
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Cryptopatches (CP) are murine gut anatomical sites for generating thymus-independent intraepithelial T lymphocytes (IEL). However, it remains elusive how lympho-hematopoietic progenitor cells migrate from bone marrow (BM) into CP and differentiate into IEL. Here we show that mice reconstituted with BM-derived c-kit(+) cells express %CCL25% (%TECK%)-intrakine gene, which reduces specifically the chemotactic response to %CCL25% but not CXCL12 in the thymocytes. These mice exhibited a dramatic reduction of CP and IEL in the small %intestine% , and harbored conspicuously decreased numbers of c-kit(+) cells in the emaciated CP. In contrast, T cells in the thymic, splenic and lymph node compartments developed normally in these mice. Importantly, it was demonstrated that CD11c(+) dendritic stromal cells in CP expressed %CCL25% and c-kit(+) Lin(-) BM cells displayed vigorous chemotactic response to %CCL25%. Furthermore, RT-PCR analysis detects mRNA expression of CCR9 in the c-kit(+) Lin(-) BM cells. Thus, these results demonstrate that the %CCL25% -CCR9 pathway is essential for CP formation and the consequent appearance of IEL.
Record Date Created: 20020703
Record Date Completed: 20030520

2/7/31 (Item 31 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

13657015 PMID: 11884450
A role for CCR9 in T lymphocyte development and migration.
Uehara Shoji; Grinberg Alexander; Farber Joshua M; Love Paul E
Laboratory of Mammalian Genes and Development, National Institute of Child Health and Human Development, and Laboratory of Clinical Investigation, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20892.
Journal of immunology (Baltimore, Md. - 1950) (United States) Mar 15 2002, 168 (6) p2811-9, ISSN 0022-1767--Print Journal Code: 2985117R
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
CCR9 mediates chemotaxis in response to %CCL25% /thymus-expressed chemokine and is selectively expressed on T cells in the thymus and small %intestine% . To investigate the role of CCR9 in T cell development, the CCR9 gene was disrupted by homologous recombination. B cell development, thymic alphabeta-T cell development, and thymocyte selection appeared unimpaired in adult CCR9-deficient (CCR9(-/-)) mice. However, competitive transplantation experiments revealed that bone marrow from CCR9(-/-) mice was less efficient at repopulating the thymus of lethally irradiated Rag-1(-/-) mice than bone marrow from littermate CCR9(+/+) mice. CCR9(-/-) mice had increased numbers of peripheral gammadelta-T cells but reduced numbers of gammadeltaTCR(+) and CD8alphabeta(+)alphabetaTCR(+) intraepithelial lymphocytes in the small %intestine%. Thus, CCR9 plays an important, although not indispensable, role in regulating the development and/or migration of both alphabeta(-) and gammadelta(-) T lymphocytes.
Record Date Created: 20020308
Record Date Completed: 20020415

2/7/32 (Item 32 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

13651322 PMID: 11874561
Chemokine and cytokine expression in murine %intestinal% epithelium following Nippostrongylus brasiliensis infection.
Rosbottom Anne; Knight Pamela A; McLachlan Gerry; Thornton Elizabeth M; Wright Steven W; Miller Hugh R P; Scudamore Cheryl L
Department of Veterinary Pathology, University of Edinburgh, Easter Bush Veterinary Centre, Roslin, Midlothian, UK.
Parasite immunology (England) Feb 2002, 24 (2) p67-75, ISSN 0141-9838--Print Journal Code: 7910948
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Infection of mice with the nematode parasite Nippostrongylus brasiliensis results in a well characterized %intestinal% mastocytosis with intraepithelial migration of mucosal mast cells (MMC). The molecules mediating this response are unknown. We examined expression of several putative mast cell chemoattractants in %intestinal% epithelium following N. brasiliensis infection. Expression of the chemokines monocyte chemoattractant protein-1 (MCP-1), macrophage inflammatory protein-1 alpha (MIP-1alpha), RANTES (regulated on activation normal T-cell expressed and secreted), fractalkine, and thymocyte expressed chemokine (%TECK%); and the cytokines stem cell factor (SCF) and transforming growth factor beta1 (TGFbeta1), was constitutive and no alteration was detected following infection. MCP-1 expression was also constitutive but at much lower levels and increased expression was detected on days 7 and 14 postinfection. Expression of MCP-1 in whole jejunum was at much higher levels than in epithelium. Constitutive expression of MCP-1, MIP-1alpha and TGFbeta1 was

also detected in cultured bone marrow-derived homologues of MMC. In an %intestinal% epithelial cell line (CMT-93), there was constitutive expression of SCF, TGFalpha1, fractalkine and MCP-1. The results show that, in vivo, epithelium is a potentially important source of mast cell chemoattractants.
Record Date Created: 20020304
Record Date Completed: 20020612

2/7/33 (Item 33 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

13618582 PMID: 11825560
Chemokines and the tissue-specific migration of lymphocytes.
Kunkel Eric J; Butcher Eugene C
Laboratory of Immunology and Vascular Biology, Department of Pathology, Stanford University School of Medicine, Stanford, CA 94305, USA. ejkunkel@cmgm.stanford.edu
Immunity (United States) Jan 2002, 16 (1) p1-4, ISSN 1074-7613--Print Journal Code: 9432918
Publishing Model Print
Document type: Journal Article; Review
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Tissue-selective trafficking of memory and effector T and B lymphocytes is mediated by unique combinations of adhesion molecules and chemokines. The discovery of several related epithelial-expressed chemokines (%TECK%/ %CCL25% in small %intestine%, CTACK/CCL27 in skin, and MEC/CCL28 in diverse mucosal sites) now highlights an important role for epithelial cells in controlling homeostatic lymphocyte trafficking, including the localization of cutaneous and %intestinal% memory T cells, and of IgA plasma cells. Constitutively expressed epithelial chemokines may help determine the character of local immune responses and contribute to the systemic organization of the immune system. (28 Refs.)
Record Date Created: 20020204
Record Date Completed: 20020221

2/7/34 (Item 34 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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13608194 PMID: 11805153
The %intestinal% chemokine thymus-expressed chemokine (%CCL25%) attracts IgA antibody-secreting cells.
Bowman Edward P; Kuklin Nelly A; Youngman Kenneth R; Lazarus Nicole H; Kunkel Eric J; Pan Junliang; Greenberg Harry B; Butcher Eugene C
Laboratory of Immunology and Vascular Biology, Department of Pathology and the Digestive Disease Center, Stanford University Medical School, Stanford, CA 94305-8444, USA.
Journal of experimental medicine (United States) Jan 21 2002, 195 (2) p269-75, ISSN 0022-1007--Print Journal Code: 2985109R
Contract/Grant No.: 5 T32 AI07290; AI; NIAID; 5R37AI121362-16; AI; NIAID; AI37832; AI; NIAID; AI47822; AI; NIAID; DK10022; DK; NIDDK; GM37734; GM; NIGMS
Publishing Model Print; Comment in J Exp Med. 2002 Jan 21;195(2) F5-8; Comment in PMID 11805155
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Immunoglobulin A (IgA) provides protection against pathogens at mucosal surfaces. Chemotactic responses have been hypothesized to target IgA plasma cells involved in mucosal immune responses. We show here that thymus-expressed chemokine (%TECK%, %CCL25%) is a potent and selective chemoattractant for IgA antibody-secreting cells (ASC), efficiently recruiting IgA-producing cells from spleen, Peyer's patches, and mesenteric lymph node. Cells secreting IgA antibody in response to rotavirus, an %intestinal% pathogen, also respond well. In contrast, IgG- and IgM-ASC respond poorly. Epithelial cells in the small %intestines%, a principal site of IgA-ASC localization and IgA production in the body, highly and selectively express %TECK%. The migration of IgA-ASC to the %intestinal% epithelial cell chemokine %TECK% may help target IgA-producing cells to the gut wall, thus helping define and segregate the %intestinal% immune response.
Record Date Created: 20020123
Record Date Completed: 20020328

2/7/35 (Item 35 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

13579789 PMID: 11781372
Rapid acquisition of tissue-specific homing phenotypes by CD4(+) T cells activated in cutaneous or mucosal lymphoid tissues.
Campbell Daniel J; Butcher Eugene C
Laboratory of Immunology and Vascular Biology, Department of Pathology, Stanford University School of Medicine, Stanford, CA 94305, USA. daniel@macampbell.com
Journal of experimental medicine (United States) Jan 7 2002, 195 (1) p135-41, ISSN 0022-1007--Print Journal Code: 2985109R
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Effector and memory T cells can be subdivided based on their ability to traffic through peripheral tissues such as inflamed skin and %intestinal% lamina propria, a property controlled by expression of 'tissue-specific' adhesion and chemoattractant receptors. However, little is known about the development of these selectively homing T cell subsets, and it is unclear whether activation in cutaneous versus %intestinal% lymphoid organs directly results in effector/memory T cells that differentially express adhesion and chemoattractant receptors targeting them to the corresponding nonlymphoid site. We define two murine CD4(+) effector/memory T cell subsets that preferentially localize in cutaneous or %intestinal% lymphoid organs by their reciprocal expression of the adhesion molecules P-selectin ligand (P-lig) and alpha 4 beta 7, respectively. We show that within 2 d of systemic immunization CD4(+) T cells activated in cutaneous lymph nodes upregulate P-lig, and downregulate alpha 4 beta 7, while those responding to antigen in %intestinal% lymph nodes selectively express high levels of alpha 4 beta 7 and acquire responsiveness to the %intestinal% chemokine thymus-expressed chemokine (%TECK%). Thus, during an immune response, local microenvironments within cutaneous and %intestinal% secondary lymphoid organs differentially direct T cell expression of these adhesion and chemoattractant receptors, targeting the resulting effector T cells to the inflamed skin or %intestinal% lamina propria.
Record Date Created: 20020108
Record Date Completed: 20020131

2/7/36 (Item 36 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

13573977 PMID: 11751956
Characterization of CCR9 expression and %CCL25% /thymus-expressed chemokine responsiveness during T cell development: CD3(high)CD69+ thymocytes and gammadeltaTCR+ thymocytes preferentially respond to %CCL25%.

Uehara Shoji; Song Kaimei; Farber Joshua M; Love Paul E
Laboratory of Mammalian Genes and Development, National Institute of Child Health and Human Development, National Institutes of Health, 9000 Rockville Pike, Bethesda, MD 20892, USA.
Journal of immunology (Baltimore, Md. - 1950) (United States) Jan 1 2002, 168 (1) p134-42, ISSN 0022-1767--Print Journal Code: 2985117R
Publishing Model Print

Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
CCR9 mediates chemotaxis of thymocytes in response to %CCL25% /thymus-expressed chemokine, and its mRNA is selectively expressed in thymus and small %intestine%, the two known sites of T lymphopoiesis. To examine the expression of CCR9 during lymphocyte development, we generated polyclonal Ab that recognizes murine CCR9. CCR9 was expressed on the majority of immature CD4+CD8+ (double-positive) thymocytes, but not on immature CD4(-)CD8(-) (double-negative) thymocytes. CCR9 was down-regulated during the transition of double-positive thymocytes to the CD4+ or CD8+ (single-positive) stage, and only a minor subset of CD8+ lymph node T cells expressed CCR9. All CCR9+ thymocyte subsets migrated in response to %CCL25% ; however, CD69+ thymocytes demonstrated enhanced %CCL25%-induced migration compared with CD69(-) thymocytes. Ab-mediated TCR stimulation also enhanced %CCL25% responsiveness, indicating that %CCL25%-induced thymocyte migration is augmented by TCR signaling. Approximately one-half of all gammadeltaTCR+ thymocytes and peripheral gammadeltaTCR+ T cells expressed CCR9 on their surface, and these cells migrated in response to %CCL25%. These findings suggest that CCR9 may play an important role in the development and trafficking of both alphabetaTCR+ and gammadeltaTCR+ T cells.
Record Date Created: 20011225
Record Date Completed: 20020111

2/7/37 (Item 37 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13454475 PMID: 11696193
Age-related changes in CCR9+ circulating lymphocytes: are CCR9+ naive T cells recent thymic emigrants?
Olaussen R W; Farstad I N; Brandtzaeg P; Rugtveit J
Laboratory for Immunohistochemistry and Immunopathology (LIIPAT), Institute of Pathology, University of Oslo, Rikshospitalet, N-0027 Oslo, Norway.
Scandinavian journal of immunology (England) Nov 2001, 54 (5) p435-9 , ISSN 0300-9475--Print Journal Code: 0323767
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The chemokine receptor CCR9 is reported to be predominantly expressed by thymocytes as well as by circulating gut-homing and resident T cells in the small %intestinal% mucosa. Its ligand thymus-expressed chemokine (%TECK%) is produced by thymic and small %intestinal% epithelium. Here we report that the proportion of circulating CCR9+ naive T cells (mostly CD4+) declines with age, from approximately 15% of all T cells at birth to around 1% in adults. The proportion of CCR9+ T cells lacking the classical gut-homing receptor alpha4beta7, was much higher in children than in adults. Therefore, circulating CD3+CCR9+CD45RA+ cells have most likely left the thymus quite recently. This notion was supported by the small number of CCR9+ naive T cells which was present shortly after thymectomy. Establishing a phenotypic marker for recent thymic emigrants might provide a powerful tool in the clinical assessment and follow-up after cancer chemotherapy, hematopoietic stem cell transplantation, and during antiretroviral treatment of human immunodeficiency virus (HIV)-infected patients.
Record Date Created: 20011106
Record Date Completed: 20011205

2/7/38 (Item 38 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13435758 PMID: 11675330
Mice lacking the CCR9 CC-chemokine receptor show a mild impairment of early T- and B-cell development and a reduction in T-cell receptor gammadelta(+) gut intraepithelial lymphocytes.
Wurbel M A; Malissen M; Guy-Grand D; Meffre E; Nussenzweig M C; Richelme M; Carrier A; Malissen B
Centre d'Immunologie de Marseille-Luminy, INSERM-CNRS- Universite de la Mediterranee, Campus de Luminy, Marseille, France.
Blood (United States) Nov 1 2001, 98 (9) p2626-32, ISSN 0006-4971--Print Journal Code: 7603509
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
CC chemokine receptor (CCR) 9, the receptor for the CC-chemokine %CCL25% /thymus-expressed chemokine (%TECK%), is mainly expressed by thymocytes and by intraepithelial (IEL) and lamina propria lymphocytes of the small %intestine% . To study the biologic role of CCR9, a mouse strain was generated in which the CCR9 gene was deleted. In spite of the high level of CCR9 found in double- and single-positive thymocytes and of the expression of its corresponding ligand on thymic stromal cells, CCR9 deletion had no major effect on intrathymic T-cell development. It was noted that there was only a one-day lag in the appearance of double-positive cells during fetal ontogeny in CCR9(-/-) thymi. When tested in chemotaxis assay, thymocytes isolated from CCR9(-/-) mice failed to respond to %TECK%/%CCL25%. Taken together, these results suggest that in thymocytes, CCR9 is the only physiologic receptor for %TECK%/%CCL25% , and that it is dispensable for proper T-cell development. Bone marrow pre-pro-B cells migrate in response to %TECK%/%CCL25% , but more mature B cells do not. Consistent with this observation, it was shown that there are fewer pre-pro-B cells in CCR9(-/-) mice than in wild-type mice. However, this diminution does not appear to have a detectable effect on the generation of a normal complement of mature B cells. Finally, it was shown that in the small %intestine% of CCR9-deficient mice, the intraepithelial T-cell-to-epithelial cell ratio is decreased, an observation that can be accounted for by a marked diminution of the T-cell receptor gammadelta(+) compartment.
Record Date Created: 20011024
Record Date Completed: 20011207

2/7/39 (Item 39 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13329973 PMID: 11487533
CCR9-positive lymphocytes and thymus-expressed chemokine distinguish small %bowel% from colonic %Crohn%'s disease.
Papadakis K A; Prehn J; Moreno S T; Cheng L; Kouroumalis E A; Deem R; Breaverman T; Ponath P D; Andrew D P; Green P H; Hodge M R; Binder S W; Targan S R
Department of Medicine, Division of Gastroenterology and Inflammatory Bowel Disease Center, Cedars-Sinai Medical Center, UCLA School of Medicine, 8700 Beverly Boulevard, Los Angeles, CA 90048, USA. papadakisk@cshs.org
Gastroenterology (United States) Aug 2001, 121 (2) p246-54, ISSN 0016-5085--Print Journal Code: 0374630
Contract/Grant No.: DK-46763; DK; NIDDK; DK-56328; DK; NIDDK
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
BACKGROUND & AIMS: Thymus-expressed chemokine (%TECK%) or %CCL25%) is selectively expressed in the small %bowel% (SB), where lamina propria lymphocytes (LPL) and intraepithelial leukocyte expressing the cognate chemokine receptor CCR9 predominate. We characterize the role of %TECK% and CCR9-expresing lymphocytes in small %intestinal% %Crohn%'s disease. METHODS: CCR9 expression on lymphocytes from lamina propria, mesenteric lymph node, and peripheral blood was analyzed by flow cytometry and by Northern blotting for LPL. %TECK% expression was analyzed in inflamed SB and %colon% by reverse-transcription polymerase chain reaction and immunohistochemistry. RESULTS: The fraction of CCR9(+) T cells in inflamed

SB was significantly lower than in uninvolved SB mucosa. In contrast, in peripheral blood lymphocytes, CCR9(+) lymphocytes were markedly elevated in patients with small %bowel% %Crohn% 's or celiac disease, but not in patients with purely colonic %Crohn%'s. Also, %TECK% expression is altered in inflamed small %bowel% , being intensely expressed in a patchy distribution in crypt epithelial cells in proximity to lymphocytic infiltrates. %TECK% is not expressed in either normal or inflamed %colon%. CONCLUSIONS: In SB immune-mediated diseases, there is repartitioning of CCR9(+) lymphocytes between SB and blood and an altered pattern of %TECK% expression in SB %Crohn%'s. The %TECK%/CCR9 ligand/receptor pair may play an important role in the pathogenesis of SB %Crohn%'s disease.
Record Date Created: 20010806
Record Date Completed: 20010830

2/7/40 (Item 40 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12911051 PMID: 11046037
The role of thymus-expressed chemokine and its receptor CCR9 on lymphocytes in the regional specialization of the mucosal immune system.
Papadakis K A; Prehn J; Nelson V; Cheng L; Binder S W; Ponath P D; Andrew D P; Targan S R
Department of Medicine, Division of Gastroenterology and Inflammatory Bowel Disease Center, Cedars-Sinai Medical Center, University of California, Los Angeles School of Medicine, Los Angeles, CA 90048, USA. Papadakisk@cshs.org
Journal of immunology (Baltimore, Md. - 1950) (UNITED STATES) Nov 1 2000, 165 (9) p5069-76, ISSN 0022-1767--Print Journal Code: 2985117R
Contract/Grant No.: DK-46763; DK; NIDDK; DK-56328; DK; NIDDK
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Chemokines play an important role in the migration of leukocytes at sites of inflammation, and some constitutively expressed chemokines may direct lymphocyte trafficking within lymphoid organs and peripheral tissues. Thymus-expressed chemokine (%TECK% or Ckbeta-15/%CCL25%), which signals through the chemokine receptor CCR9, is constitutively expressed in the thymus and small %intestine% but not %colon%, and chemoattracts a small fraction of PBLs that coexpress the integrin alpha(4)beta(7). Here we show that %TECK% is expressed in the human small %bowel% but not %colon% by endothelial cells and a subset of cells in %intestinal% crypts and lamina propria. CCR9 is expressed in the majority of freshly isolated small %bowel% lamina propria mononuclear cells (LPMC) and at significantly higher levels compared with colonic LPMC or PBL. %TECK% was selectively chemotactic for small %bowel% but not colonic LPMC in vitro. The %TECK% -induced chemotaxis was sensitive to pertussis toxin and partially inhibited by Abs to CCR9. %TECK% attracts predominantly the T cell fraction of small %bowel% LPMC, whereas sorted CD3(+)CCR9(+) and CD3(+)CCR9(-) lymphocytes produce similar Th1 or Th2 cytokines at the single cell level. Collectively, our data suggest that the selective expression of %TECK% in the small %bowel% underlie the homing of CCR9(+) %intestinal% memory T cells to the small %bowel% rather than to the %colon%. This regional specialization implies a segregation of small %intestinal% from colonic immune responses.
Record Date Created: 20001103
Record Date Completed: 20001130

2/7/41 (Item 41 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12852423 PMID: 10974041
Lymphocyte CC chemokine receptor 9 and epithelial thymus-expressed chemokine (%TECK%) expression distinguish the small %intestinal% immune compartment: Epithelial expression of tissue-specific chemokines as an organizing principle in regional immunity.
Kunkel E J; Campbell J J; Haraldsen G; Pan J; Boisvert J; Roberts A I; Ebert E C; Vierra M A; Goodman S B; Genovese M C; Wardlaw A J; Greenberg H B; Parker C M; Butcher E C; Andrew D P; Agace W W
Laboratory of Immunology and Vascular Biology, Department of Pathology, Stanford University School of Medicine, Stanford, California 94305, USA.
Journal of experimental medicine (UNITED STATES) Sep 4 2000, 192 (5) p761-8, ISSN 0022-1007--Print Journal Code: 2985109R
Contract/Grant No.: 5T32AI07290; AI; NIAID; AI47822; AI; NIAID; GM37734; GM; NIGMS; +
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The immune system has evolved specialized cellular and molecular mechanisms for targeting and regulating immune responses at epithelial surfaces. Here we show that small %intestinal% intraepithelial lymphocytes and lamina propria lymphocytes migrate to thymus-expressed chemokine (%TECK%). This attraction is mediated by CC chemokine receptor (CCR)9, a chemoattractant receptor expressed at high levels by essentially all CD4(+) and CD8(+) T lymphocytes in the small %intestine%. Only a small subset of lymphocytes in the %colon% are CCR9(+), and lymphocytes from other tissues including tonsils, lung, inflamed liver, normal or inflamed skin, inflamed synovium and synovial fluid, breast milk, and seminal fluid are universally CCR9(-). %TECK% expression is also restricted to the small %intestine%: immunohistochemistry reveals that intense anti-%TECK% reactivity characterizes crypt epithelium in the jejunum and ileum, but not in other epithelia of the digestive tract (including stomach and %colon%), skin, lung, or salivary gland. These results imply a restricted role for lymphocyte CCR9 and its ligand %TECK% in the small %intestine%, and provide the first evidence for distinctive mechanisms of lymphocyte recruitment that may permit functional specialization of immune responses in different segments of the %gastrointestinal% tract. Selective expression of chemokines by differentiated epithelium may represent an important mechanism for targeting and specialization of immune responses.
Record Date Created: 20000928
Record Date Completed: 20000928

2/7/42 (Item 42 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12581633 PMID: 10602049
The chemokine %TECK% is expressed by thymic and %intestinal% epithelial cells and attracts double- and single-positive thymocytes expressing the %TECK% receptor CCR9.
Wurbel M A; Philippe J M; Nguyen C; Victorero G; Freeman T; Wooding P; Miazek A; Mattei M G; Malissen M; Jordan B R; Malissen B; Carrier A; Naquet P
Centre d'Immunologie INSERM-CNRS de Marseille-Luminy, Marseille, France.
European journal of immunology (GERMANY) Jan 2000, 30 (1) p262-71, ISSN 0014-2980--Print Journal Code: 1273201
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Chemokines are key regulators of migration in lymphoid tissues. In the thymus, maturing thymocytes move from the outer capsule to the inner medulla and thereby interact with different types of stromal cells that control their maturation and selection. In the process of searching for molecules specifically expressed at different stages of mouse thymic differentiation, we have characterized the cDNA coding for the thymus-expressed chemokine (%TECK%) and its receptor CCR9. The %TECK% receptor gene was isolated and shown to be localized on the mouse chromosome 9F1-F4. Thymic dendritic cells have been initially thought to be a prevalent source of %TECK%. In contrast, our results indicate that thymic epithelial cells constitute the predominant source of %TECK%. Consistent

with the latter distribution, the %TECK% receptor is highly expressed by double-positive thymocytes, and %TECK% can chemoattract both double-positive and single-positive thymocytes. The %TECK% transcript is also abundantly expressed in the epithelial cells lining the small %intestine%. In conclusion, the interplay of %TECK% and its receptor CCR9 is likely to have a significant role in the recruitment of developing thymocytes to discrete compartments of the thymus.
Record Date Created: 20000124
Record Date Completed: 20000124

2/7/43 (Item 43 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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12104284 PMID: 10544196
Human G protein-coupled receptor GPR-9-6/CC chemokine receptor 9 is selectively expressed on %intestinal% homing T lymphocytes, mucosal lymphocytes, and thymocytes and is required for thymus-expressed chemokine-mediated chemotaxis.
Zabel B A; Agace W W; Campbell J J; Heath H M; Parent D; Roberts A I; Ebert E C; Kassam N; Qin S; Zovko M; LaRosa G J; Yang L L; Soler D; Butcher E C; Ponath P D; Parker C M; Andrew D P
LeukoSite, Inc., Cambridge, Massachusetts 02142, USA.
Journal of experimental medicine (UNITED STATES) Nov 1 1999, 190 (9) p1241-56, ISSN 0022-1007--Print Journal Code: 2985109R
Contract/Grant No.: AI37832; AI; NIAID; DK42166; DK; NIDDK; DK52978; DK; NIDDK; +
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
%TECK% (thymus-expressed chemokine), a recently described CC chemokine expressed in thymus and small %intestine%, was found to mediate chemotaxis of human G protein-coupled receptor GPR-9-6/L1.2 transfectants. This activity was blocked by anti-GPR-9-6 monoclonal antibody (mAb) 3C3. GPR-9-6 is expressed on a subset of memory alpha4beta7(high) %intestinal% trafficking CD4 and CD8 lymphocytes. In addition, all %intestinal% lamina propria and intraepithelial lymphocytes express GPR-9-6. In contrast, GPR-9-6 is not displayed on cutaneous lymphocyte antigen-positive (CLA(+)) memory CD4 and CD8 lymphocytes, which traffic to skin inflammatory sites, or on other systemic alpha4beta7(-)CLA(-) memory CD4/CD8 lymphocytes. The majority of thymocytes also express GPR-9-6, but natural killer cells, monocytes, eosinophils, basophils, and neutrophils are GPR-9-6 negative. Transcripts of GPR-9-6 and %TECK% are present in both small %intestine% and thymus. Importantly, the expression profile of GPR-9-6 correlates with migration to %TECK% of blood T lymphocytes and thymocytes. As migration of these cells is blocked by anti-GPR-9-6 mAb 3C3, we conclude that GPR-9-6 is the principal chemokine receptor for %TECK% . In agreement with the nomenclature rules for chemokine receptors, we propose the designation CCR-9 for GPR-9-6. The selective expression of %TECK% and GPR-9-6 in thymus and small %intestine% implies a dual role for GPR-9-6/CCR-9, both in T cell development and the mucosal immune response.
Record Date Created: 19991130
Record Date Completed: 19991130

2/7/44 (Item 44 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

11457275 PMID: 9285413
%TECK% : a novel CC chemokine specifically expressed by thymic dendritic cells and potentially involved in T cell development.
Vicari A P; Figueroa D J; Hedrick J A; Foster J S; Singh K P; Menon S; Copeland N G; Gilbert D J; Jenkins N A; Bacon K B; Zlotnik A
DNAX Research Institute, Palo Alto, California 94304-1104, USA.
Immunity (UNITED STATES) Aug 1997, 7 (2) p291-301, ISSN 1074-7613--Print Journal Code: 9432918
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
A novel CC chemokine was identified in the thymus of mouse and human and was designated %TECK% (thymus-expressed chemokine). %TECK% has weak homology to other CC chemokines and maps to mouse chromosome 8. Besides the thymus, mRNA encoding %TECK% was detected at substantial levels in the small %intestine% and at low levels in the liver. The source of %TECK% in the thymus was determined to be thymic dendritic cells; in contrast, bone marrow-derived dendritic cells do not express %TECK%. The murine %TECK% recombinant protein showed chemotactic activity for activated macrophages, dendritic cells, and thymocytes. We conclude that %TECK% represents a novel thymic dendritic cell-specific CC chemokine that is possibly involved in T cell development.
Record Date Created: 19970929
Record Date Completed: 19970929

2/7/45 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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19331350 BIOSIS NO.: 200600676745
Role of %CCL25%-CCR9 interaction in ovarian cancer cell metastasis and survival
AUTHOR: Johnson Erica L (Reprint); Singh Shailesh; Johnson-Holiday Crystal; Singh Udai P; Lillard James W
AUTHOR ADDRESS: Morehouse Sch Med, Atlanta, GA 30310 USA**USA
JOURNAL: Journal of Immunology 176 (Suppl. S): pS271 APR 1 2006 2006
CONFERENCE/MEETING: Annual Meeting of the American-Association-of-Immunologists Boston, MA, USA May 12 -16, 2006; 20060512
SPONSOR: Amer Assoc Immunologists
ISSN: 0022-1767
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

2/7/46 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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19330236 BIOSIS NO.: 200600675631
Murine chemokine %CCL25% gene inactivation results in TCRgd plus compartment deficiency in the small %intestine% and its accumulation in secondary lymphoid organs
AUTHOR: Wurbel Marc-Andre (Reprint); Guy-Grand Delphine; Campbell James J; Malissen Bernard
AUTHOR ADDRESS: Childrens Hosp, Joint Program Transfus Med, Boston, MA 02115 USA**USA
JOURNAL: Journal of Immunology 176 (Suppl. S): pS31 APR 1 2006 2006
CONFERENCE/MEETING: Annual Meeting of the American-Association-of-Immunologists Boston, MA, USA May 12 -16, 2006; 20060512
SPONSOR: Amer Assoc Immunologists
ISSN: 0022-1767
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/47 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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19248908 BIOSIS NO.: 200600594303
%CCL25%-CCR9 axis'role in ovarian cancer cell metastasis and survival.

AUTHOR: Johnson Erica L (Reprint); Singh Shailesh; Johnson-Holiday Crystal; Singh Udai P; Partridge Edward E; Datta Milton W; Lillard James W
AUTHOR ADDRESS: Morehouse Sch Med, Atlanta, GA 30310 USA**USA
JOURNAL: Proceedings of the American Association for Cancer Research Annual Meeting 47 p70 APR 2006 2006
CONFERENCE/MEETING: 97th Annual Meeting of the American-Association-for-Cancer-Research (AACR) Washington, DC, USA April 01 -05, 2006; 20060401
SPONSOR: Amer Assoc Canc Res
ISSN: 0197-016X
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/48 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19215272 BIOSIS NO.: 200600560667
Liver dendritic cells in primary sclerosing cholangitis (PSC) are unable to imprint mucosal adhesion molecules in primed lymphocytes without exogenous retinoic acid
AUTHOR: Eksteen B (Reprint); Curbishley S M; Lai W K; Adams D H
AUTHOR ADDRESS: Univ Birmingham, Liver Res Grp, Birmingham, W Midlands, UK **UK
JOURNAL: Journal of Hepatology 44 (Suppl. 2): pS10 2006 2006
CONFERENCE/MEETING: 41st Annual Meeting of the European-Association-for-the-Study-of-the-Liver Vienna, AUSTRIA April 26 -30, 2006; 20060426
SPONSOR: European Assoc Study Liver
ISSN: 0168-8278
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

2/7/49 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19159334 BIOSIS NO.: 200600504729
A critical role for beta 7-integrins but not CCR9 or %CCL25% in the pathogenesis of experimental ileitis
AUTHOR: Apostolaki Maria; Papadakis Konstantinos A; Manoloukos Menelaos; Wurbel Mar-Andre; Avanesyan Armine; Saruta Masayuki; Kontoyiannis Dimitris L; Malissen Bernard; Kollias George
JOURNAL: Gastroenterology 130 (4, Suppl. 2): pA550 APR 2006 2006
CONFERENCE/MEETING: Digestive Disease Week Meeting/107th Annual Meeting of the American-Gastroenterological-Association Los Angeles, CA, USA May 19 -24, 2006; 20060519
SPONSOR: Amer Gastroenterol Assoc Inst
ISSN: 0016-5085
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background and Aims: The chemokine %CCL25% and its receptor CCR9 have been reported to play an important role in small %bowel% immunity. The upregulated expression of %CCL25% and the increased frequency of activated peripheral blood CCR9+ T cells in SB %Crohn%'s disease (CD) implicate %CCL25%/CCR9 in the pathogenesis,of the disease. The aim of our study was to determine the role of %CCL25% and CCR9, as,well as of the integrin.beta 7, in the pathogenesis of ileitis in the TNF Delta ARE model of %Crohn%'s-like disease. Methods: %CCL25% mRNA and protein expression in the small %bowel% (SB) of TNF Delta ARE and wild-type mice were assessed by real-time PCR and ELISA, respectively. The effect of CCR9 and beta 7 integrin in the course of ileitis was evaluated by backcrossing the TNF Delta ARE mice with CCR94- and 07 integrin-/- mice, whereas a neutralizing anti-%CCL25% Ab was used to assess the effect of %CCL25% in the course of experimental ileitis. Results: %CCL25% was upregulated early (1month) but not late (4 months) during the development of the disease in the TNF Delta ARE mice compared to wild-type mice. Administration of anti-%CCL25% Ab at 5 mg/kg twice per week for 10 weeks, starting at 3 weeks of age, to TNF Delta ARE mice led to no significant improvement of the ileitis compared to isotype control-treated mice. Backcrossing of TNF Delta ARE mice with CCR9-/- mice led to similar ileitis development compared to CCR9-sufficient mice. In marked contrast, backcrossing TNF Delta ARE mice with 07 integrin-/- mice led to significant amelioration of the disease. Conclusion: beta 7 integrin is critically involved in the pathogenesis of experimental ileitis, whereas %CCL25% and CCR9 appear to play a dispensable role. %CCL25% may be involved in the early, but not late stages of the disease.

2/7/50 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18999919 BIOSIS NO.: 200600345314
Murine zinc deficiency alters lymphocyte phenotypes and %CCL25% expression in the %colon%
AUTHOR: Herrlinger-Garcia Kelli (Reprint); Silvestre Justin; Knutson Mitchell D; Litherland Sally A; Cousins Robert J; Langkamp-Henken Bobbi
AUTHOR ADDRESS: Univ Florida, Gainesville, FL 32611 USA**USA
JOURNAL: FASEB Journal 20 (4, Part 1): pA603 MAR 6 2006 2006
CONFERENCE/MEETING: Experimental Biology 2006 Meeting San Francisco, CA, USA April 01 -05, 2006; 20060401
SPONSOR: Amer Assoc Anatomists
Amer Physiol Soc
Amer Soc Biochem & Mol Biol
Amer Soc Investigat Pathol
Amer Soc Nutr
Amer Soc Pharmacol & Expt Therapeut
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Zinc deficiency alters T(H)1/T(H)2 cytokine balance. Although these cytokine changes may alter lymphocyte phenotypic distribution or homing, no studies have examined this outcome. Balb/c mice (4 wk old) were fed a zinc-deficient (ZD, <1 mg Zn/kg), zinc-adequate (ZA, 27 mg Zn/kg), or pair-fed (PF) diet for 9 wk. Percent baseline weight did not differ among the 3 groups in this T(H)2-dominant mouse strain; therefore, PF was dropped from further analyses. Serum zinc (ug/ml) decreased with progressive zinc deficiency and at wk 9 was 0.7 +/- 0.1 (ZA) vs. 0.3 +/- 0.2 (ZD, mean +/- SEM, P = 0.001). Colonic intraepithelial lymphocyte (cIEL) phenotypes were evaluated at 3, 6, and 9 wk using flow cytometry and anti-CD3, CD8 beta, TCR gamma delta and CD4 antibodies. ZD CD3(+) cIEL (T cells) normalized to the ZA group increased with progressive zinc deficiency (P = 0.06), and the percentage of CD3(+) T cells was higher at wk 9 in ZD vs. ZA (43 +/- 4% vs. 29 +/- 3%, P = 0.04). CD3(+)CD8 beta+TCR gamma delta(-) cIELs were elevated at all time points in ZD mice (P = 0.008) and may account for the T-cell increase. At wk 9, colonic inflammatory cytokines and receptors were measured using a microarray followed by qRT-PCR. Microarray analysis indicated that %CCL25% (%TECK%) was overexpressed in ZA %colon% whereas IL-18 was overexpressed in ZD. qRT-PCR confirmed that normalized %CCL25% mRNA levels were different between diet groups, whereas IL-18 mRNA levels were unchanged. However, IL-18 transcript levels positively correlated with the percentage of ZA and ZD CD3(+)CD3(+)TCR gamma delta(-) cells. These data suggest that ZD alters colonic lymphocyte phenotypic distribution and a chemokine ligand important in lymphocyte homing.

2/7/51 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18998065 BIOSIS NO.: 200600343460
Role of chemokines in lymphocyte trafficking to the %intestine% in chronic murine ileitis
AUTHOR: Schwartz Marc Andre (Reprint); Kivera-Nieves Jesus; Ley Klaus
AUTHOR ADDRESS: Univ Virginia, Dept Biomed Engn, Charlottesville, VA 22903 USA**USA
JOURNAL: FASEB Journal 20 (4, Part 1): pA203 MAR 6 2006 2006
CONFERENCE/MEETING: Experimental Biology 2006 Meeting San Francisco, CA, USA April 01 -05, 2006; 20060401
SPONSOR: Amer Assoc Anatomists
Amer Physiol Soc
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Amer Soc Investigat Pathol
Amer Soc Nutr
Amer Soc Pharmacol & Expt Therapeut
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The recirculating lymphocytes responsible for maintaining chronic inflammation in %Crohn%'s disease traffic to the small %intestine% using an array of adhesion molecules and chemokines. Tissue-specific trafficking to the %intestine% under normal physiologic conditions is controlled by the integrin alpha(4)beta(7) and the chemokine receptor CCR9 expressed on lymphocytes recognizing their ligands MAdCAM-1 and %CCL25% (%TECK%), respectively, on endothelial cells in the %intestinal% microvasculature. We investigated the role of several chemokine receptors in the pathogenesis of chronic murine ileitis using RT-PCR. At 10 weeks of age, when inflammation has developed but has not yet reached peak severity, expression of CCR6 and CCR7 in ilea was found to be 2-fold and 4-fold higher, respectively, in mice with ileitis relative to age-matched controls. By 40 weeks, when chronic inflammation is well-established, CCR7 showed no further increase in expression, but CCR6 expression had increased to 10-fold greater than controls. CCR2, CCR5, CXCR2, CXCR3, and CXCR4 were also expressed at higher levels in mice with ileitis at 40 weeks. These data suggest that lymphocytes in mice with well-established inflammation produce several inflammatory chemokine receptors that contribute to ileitis, but disease initiation involves dysregulation of a smaller set of tissue-specific recruitment pathways.

2/7/52 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18866142 BIOSIS NO.: 200600211537
The chemokine/receptor pair %CCL25%/CCR9 participates in the inductive stages of spontaneous murine chronic ileitis.
AUTHOR: Rivera-Nieves Jesus; Bamias Giorgos; Knight Robert F; Ivashkina Natalia; Hoang Sharon; Wei Zheng; Love Paul E; Kollias Giorgos; Opperman Martin; Ley Klaus; Cominelli Fabio
JOURNAL: Gastroenterology 128 (4, Suppl. 2): pA508 APR 2005 2005
CONFERENCE/MEETING: Annual Meeting of the American-Gastroenterological-Association/Digestive-Disease-Week Chicago, IL, USA May 14 -19, 2005; 20050514
SPONSOR: Amer Gastroenterol Assoc
ISSN: 0016-5085
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: %Crohn%'s disease (CD) affects the small %intestine% (chronic ileitis) in at least two thirds of cases. Recently, the chemokine %CCL25% was shown to be restrictively expressed in the small %intestine%, but its role in chronic ileitis has yet to be elucidated. Aim: The aim of this study was to investigate the role of %CCL25% and its receptor CCR9 in the development of %intestinal% inflammation in the SAMP1/YitFc (SAMP) and TNF Delta ARE murine models of chronic ileitis. Methods: Flow cytometry, immunohistochemistry (IHC) and real-time RT-PCR were used to determine tissue mRNA expression and cellular localization of CCR9 and %CCL25% during the course of the disease. Monoclonal antibodies against CCR9 or %CCL25%/%TECK% (200 mu g, Q O.D., I.P. x 3) were used to assess the effect of functional blockade of the chemokine/receptor pair on the severity of ileitis. In addition, recombinant TNF Delta ARE/CCR9-deficient mice were generated to determine whether CCR9 was required for disease development. Results: Expression of CCR9 in CD4 + and CD8 + T cells increased 3-4-fold in lamina propria, MLN and spleen cells from inflamed mice compared with controls. %CCL25% localized predominantly to the small %intestinal% crypts, and its expression decreased with disease progression in SAMP mice but not in uninflamed age-matched mice. Ileitis seventy was significantly attenuated by %CCL25% or CCR9 immunoblockade in SAMP mice. By contrast, CCR9 deficiency failed to attenuate ileitis in TNF Delta ARE/CCR9(-/-) mice. Conclusions: Increased expression of CCR9 by CD4 + and CD8 + T cells in inflamed mice and attenuation of disease after CCR9 or %CCL25% immunoblockade support a role for this chemokine/receptor pair in ileitis. Furthermore, decreased %CCL25% expression with disease progression suggests that the chemokine may be more important for induction, rather than for maintenance of disease. However, failure of CCR9 deficiency to attenuate inflammation in TNF Delta ARE mice implies that other pathways compensate for CCR9 deficiency in chronic ileitis. Thus, immunoblockade of %CCL25%/CCR9 may be efficacious for the treatment of certain subsets of patients with CD. In addition, therapeutic efficacy may be enhanced by simultaneous blockade of other %intestinal% lymphocyte homing pathways.

2/7/53 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18864653 BIOSIS NO.: 200600210048
Cc chemokine receptor 9 (ccr9) antagonist ameliorates experimental ileitis and colitis
AUTHOR: Wei Zheng; Ertl Linda; Baumgart Trageen; Rubas Wemer; Hor Sok-Ying ; Wright J J Kim; Howard Maureen; Schall Thomas; Keshav Satish
JOURNAL: Gastroenterology 128 (4, Suppl. 2): pA204-A205 APR 2005 2005
CONFERENCE/MEETING: Annual Meeting of the American-Gastroenterological-Association/Digestive-Disease-Week Chicago, IL, USA May 14 -19, 2005; 20050514
SPONSOR: Amer Gastroenterol Assoc
ISSN: 0016-5085
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: %CCL25% (also %TECK% or thymus-expressed chemokine) is highly expressed in the small %intestine% and thymic stroma. It stimulates chemotaxis by binding to its cognate receptor CCR9, which is expressed on double-positive thymocytes and a small minority of peripheral blood lymphocytes. Almost all lymphocytes in the small %intestine% and approximately 30% in the large %intestine% are CCR9 positive, suggesting that this chemokine receptor and ligand pair is critically important in localizing lymphocytes within the %gastrointestinal% tract in health and disease. Aims: To determine if pharmacological blockade of CCR9 could ameliorate experimental inflammatory %bowel% disease.Methods: CCX282 (Traficet-EN) is a highly selective and potent (IC50 1nM) small molecule inhibitor of CCR9-mediated chemotaxis, which was developed to achieve excellent in vivo pharmacokinetic properties and oral bioavailability and was entirely free from toxicity in a comprehensive range of pre-clinical measurements. CCX282 was administered by subcutaneous injection in two murine models of inflammatory %bowel% disease. The effect on inflammation in the small %bowel% was determined using a well-established model of ileal %Crohn%'s disease, the TNFAARE mouse, in which regulatory elements of the TNFa gene have been deleted. The effect on inflammation in the large %intestine% was determined using a model of ulcerative colitis caused by transgenic disruption of the MDR1a gene. An unrelated chemokine receptor antagonist and vehicle only were administered in parallel as controls.Results:

Blockade of CCR9 significantly ameliorated the severity of ileitis and colitis in the animal models, as determined by clinical and histopathological measurements, No adverse effects of CCX282 treatment were observed, and the control treatments were ineffective.Conclusion: Blockade of CCR9-mediated lymphocyte recruitment has efficacy in animal models and the data raise the prospect of a safe and effective oral therapy, with a novel mode of action, for human inflammatory %bowel% diseases.

2/7/54 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18838281 BIOSIS NO.: 200600183676
In vitro cultured allogeneic cytotoxic T cells mediate hematopoietic GVHD without gut GVHD despite expression of functional LPAM (alpha(4)beta(7) integrin).
AUTHOR: Kalpatthi Ram (Reprint); Nicol Kathleen; Hendey Lindsay; Boyer Michael W
AUTHOR ADDRESS: Childrens Hosp, Columbus, OH 43205 USA**USA
JOURNAL: Blood 106 (11, Part 1): p379A-380A NOV 16 2005 2005
CONFERENCE/MEETING: 47th Annual Meeting of the American-Society-of-Hematology Atlanta, GA, USA December 10 -13, 2005; 20051210
SPONSOR: Amer Soc Hematol
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Graft-versus-host disease (GVHD) remains a significant complication of allogeneic bone marrow transplantation. We and others have shown that in vitro cultured CD8 cytotoxic T cells (CTL) have attenuated GVHD compared to naive cells, while retaining GVL activity. It has been shown that expression of alpha(4)beta(7) integrin on CD8 T cells is important for gut homing specificity in GVHD. Recently, retinoic acid (RA) has been shown to upregulate a,p,expression on naive T cells. Thus, we hypothesize that in vitro cultured CTL without RA lack the ability to cause GVHD in part due to deficient alpha(4)beta(7) expression. We used an established marine GVHD model in which spleen and lymph node cells from B6SJL mice were stimulated with splenocytes from DBA mice and were restimulated on day 7. Cultures were supplemented with IL-2 & IL-7 with and without addition of RA (100nm) on day 2. Day 14 comparison of CTL with and without RA revealed comparable CD4 (1.7% vs 0.7% respectively) and CD8 populations (96% vs 97% respectively). Phenotyping of CTL with and without RA showed CD8 alpha(4)beta(7) expression of 58% and 0.8% and CD8 CCR9 53% and 10% respectively. In vitro cytotoxicity was comparable between CTL with and without RA: 51% vs 41% at an effector target ratio of 10: 1 (n=3, p=0.3). Both CTL groups had comparable in vitro migration towards to SDF, IP-10 & MIP-3 alpha (p = ns). However RA treated CTL had increased migration towards %TECK% (chemokine expressed in small %intestine%); 17.3% vs 4.6% (n = 4, p = 0.01) and decreased migration towards TARC (chemokine expressed in skin); 2% vs 13% (n = 4, p = 0.03). For in vivo homing, 10(7) labeled cells from each CTL with (CFSE) and without RA (TRITC) were coinjected intravenously and mice were sacrificed 16 hours later for analysis. RA treated CTL had increased homing to peyer's patch and MLN compared to CTL without RA. [Homing index (CTLRA/CTL) 2.3 and 2.5 respectively]. This finding is exaggerated in the radiated host [Homing index (CTLRA/CTL) 15 for PIP and 11 for MLN]. CTL generated with or without RA (5 x 10(6) cells each) were injected intravenously in to irradiated (600 Rads) B6D2F1 recipients (3 groups; Radiation control, CTL with and without RA). Mice were followed for clinical GVHD scores and sacrificed when moribund. CBC and histopathologic GVHD scores (Liver, skin, lung, small and large %intestines%) were obtained. Both CTL groups developed lethal bone marrow (BM) aplasia around day 24 as compared to radiation control group; however, clinical and histopathologic GVHD scores were similar in all groups (Table 1). Our data demonstrate that both CTL with and without RA cause a lethal hematopoietic graft versus host reaction. Despite high alpha(4)beta(7) and CCR9 expression, significant in vitro migration to %TECK% and in vivo homing to gut associated lymphoid tissues, RA treated CTL did not cause significant GVHD in gut, liver or skin. This suggests that defective gut homing alone may not be sufficient to explain the attenuated GVHD from cultured CTL. Future studies are planned to confirm these findings in other GVHD models and to elucidate the mechanisms of attenuated GVHD from cultured CTL.

2/7/55 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18739736 BIOSIS NO.: 200600085131
Interactions of mucosal lymphocytes with the extracellular matrix (ECM) component fibronectin (FN)
AUTHOR: Dotan Iris; Vigodman Sharon; Broitman Lena; Tulchinsky Hagn; Abram Talia; Maharashak Nitsan; Zhanin-Zorov Alexandra; Hecht Iris; Halpern Zamir; Lider Ofer
JOURNAL: Gastroenterology 126 (4, Suppl. 2): pA424 APR 2004 2004
CONFERENCE/MEETING: Digestive Disease Week/105th Annual Meeting of the American-Gastroenterological-Association New Orleans, LA, USA May 16 -20, 2004; 20040516
SPONSOR: Amer Gastroenterol Assoc
ISSN: 0016-5085
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: Mucosal lymphocytes traverse trough the ECM to the %intestinal% lamina propria where they exert their effector functions and undergo activation-induced cell death (apoptosis). In inflammatory %bowel% diseases (IBD) mucosal lymphocyte migration is increased and apoptosis decreases. The effects that the ECM may have on mucosal lymphocytes functions have not been explored. Aim: To investigate the interactions between mucosal lymphocytes and the ECM in homeostasis and inflammation. Methods: Mononuclear cells (MC) were isolated from lamina propria (LP), mesenteric lymph nodes (MLN) and peripheral blood (PB) of IBD and non-IBD patients undergoing %bowel% surgery. phenotype was assessed by flow cytometry. Cell migration was assessed using transwells coated with the ECM component fibronectin (FN) where (51)[Cr]-labeled MC were placed in the upper, and the chemokines SDF-1 alpha, MIP-3 alpha, IP-10 and %TECK% in the lower chamber. Migration after 3 hours was detected by counting the radioactivity in the lower chamber. Spontaneous and stimulated (by anti-CD3, Fas and FasL in the presence or absence of FN) apoptosis was assessed by annexin V/propridium iodide staining and flow cytometry Results: MLN and PB MC differed from autologous Up MC in CD3 (60.1% +/- 3.0 vs. 33.9% +/- 63, p = 0.005), CD4 (70.5% +/- 2.5 vs 52.1% +/- 7.8, p = 0.003), CD45RA + (53.4% +/- 3.3 vs, 20.8 +/- 2.9, p<0.0001) and CD95 expression (54.1% +/- 5.1 vs. 87 +/- 2.4, p<0.0001) Normal MLN and PB MC migrated to SDF-1 alpha (2 fold increase) while LP MC did not migrate to any of the chemokines tested. In %Crohn%'s disease (CD) a higher (2-fold increase) migration of LP and MLN MC to SDF-1 alpha. was noted. MLN and PB MC had <10% spontaneous apoptosis, were resistant to Fas-mediated apoptosis and had a 5-fold increase in apoptosis after stimulation with anti-CD3. In contrast, 15-40% of UP MC underwent spontaneous apoptosis, which doubled following Fas engagement and was not influenced by stimulation with anti-CD3. Exposure to FasL in the presence of FN increased apoptosis by 20% in LP MC but not in MLN or PB MC. IBD LP MC had 20% lower spontaneous apoptosis. Conclusions: LP differ from MLN and PB MC by phenotype, migration and apoptotic patterns, Differences in ECM-associated apoptosis suggest that the microenvironment regulates apoptosis in distinct mucosal lymphocyte subsets. IBD Up MC have increased migration to SDF-1 alpha and decreased apoptosis in the presence of FN suggesting a mechanism to uncontrolled inflammation in the LP.

2/7/56 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18727008 BIOSIS NO.: 200600072403
CCR9 directs plasmacytoid dendritic cells to the %intestinal% lamina propria
AUTHOR: Wendland M (Reprint); Pabst O; Czeloth N; Foerster R
JOURNAL: Immunobiology 210 (6-8): p613-614 2005 2005
CONFERENCE/MEETING: Joint Annual Meeting of the German and Scandinavian Societies of Immunology Kiel, GERMANY September 21 -24, 2005; 20050921
SPONSOR: German Soc Immunol
Scandinavian Soc Immunol
ISSN: 0171-2985
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/57 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18714184 BIOSIS NO.: 200600059579
SCID mice transplanted with human gut as a pre-clinical model for studying the migration of human lymphocytes to the small %intestine%
AUTHOR: Guenther C (Reprint); Schwaerzler C; Kund J; Carballido J; Hinteregger S; Fassl S; Biedermann T; Carballido J
AUTHOR ADDRESS: Novartis Inst Biomed Res, Vienna, Austria**Austria
JOURNAL: Journal of Investigative Dermatology 125 (3, Suppl. S): pA43 SEP 2005 2005
CONFERENCE/MEETING: 35th Annual Meeting of the European-Society-for-Dermatological-Research Tubingen, GERMANY September 22 -24, 2005; 20050922
SPONSOR: European Soc Dermatol Res
Centocor Schering Plough
Galderma
Novartis
Chanel
Hermal Germany
Nat Publ Grp
Fujisawa
Intendis
Serono
ISSN: 0022-202X
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/58 (Item 14 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18680609 BIOSIS NO.: 200600026004
Annals of the New York Academy of Sciences
BOOK TITLE: Annals of the New York Academy of Sciences
AUTHOR: Weiner HL; Mayer L; Strober W
BOOK AUTHOR/EDITOR: Weiner HL (Editor); Mayer L (Editor); Strober W (Editor)
SERIES TITLE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES 1029 2004
BOOK PUBLISHER: NEW YORK ACAD SCIENCES, 2 EAST 63RD ST, NEW YORK, NY 10021 USA
CONFERENCE/MEETING: Conference on Oral Tolerance New York, NY, USA October 23 -26, 2003; 20031023
SPONSOR: New York Acad Sci
ISSN: 0077-8923_(print) ISBN: 1-57331-508-7 (H); 1-57331-509-5 (S)
DOCUMENT TYPE: Book; Meeting; Meeting Summary
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: This 421-page book, entitled 'Oral Tolerance: New Insights and Prospects for Clinical Application' is volume 1029 in the series Annals of the New York Academy of Sciences' and is the result of a conference entitled 'Oral Tolerance: Mechanism and Applications', which was held in the new York Academy of Sciences in New York City in October 2003. Oral tolerance is a long-recognized mechanism of inducing immune tolerance and has become an important area of investigation in terms of basic research and clinical applications. Since the last conference on oral tolerance there has been a huge improvement in our understanding of potential mechanisms of oral tolerance through the use of mouse models. There has been a greater appreciation for the existence of novel regulatory T cells and a clearer view has emerged of how the mucosal immune system responds. This volume brings together up-to-date knowledge in these areas and is structured into 6 parts. The book contains 61 individually-authored chapters, all written in English and each with an extensive list of references. Broad topics covered in these sections of the book include the anatomy and physiology of the mucosal immune response, the role of dendritic cells in oral tolerance, the role of regulatory T cells in oral tolerance, in vivo effectors of oral tolerance, and oral tolerance in animal disease models and human trials. The sixth and final part of the book contains a number of short papers on oral tolerance and the immune system. The book is indexed by contributor. This will be a useful resource for researcher in clinical immunology and clinical immunologists.

2/7/59 (Item 15 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18631620 BIOSIS NO.: 200510326120
Role and function of CCR9 and hypoxia in ovarian cancer cell motility and invasion
AUTHOR: Johnson Erica (Reprint); Singh Shailesh; Singh Udai P; Partridge Edward; Lillard James W Jr
AUTHOR ADDRESS: Morehouse Sch Med, Atlanta, GA 30310 USA**USA
JOURNAL: FASEB Journal 19 (4, Suppl. S, Part 1): pA943 MAR 4 2005 2005
CONFERENCE/MEETING: Experimental Biology 2005 Meeting/35th International Congress of Physiological Sciences San Diego, CA, USA March 31 -April 06, 2005; 20050331
SPONSOR: Amer Assoc Anatomists
Amer Assoc Immunologists
Amer Physiol Soc
Amer Soc Biochem & Mol Biol
Amer Soc Investigat Pathol
Amer Soc Nutr Sci
Amer Soc Pharmacol & Expt Therapeut
Int Union Physiol Sci
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Epithelial ovarian cancer is the most lethal gynecological cancer among women and its poor prognosis is mainly due to metastasis. Chemokines have been shownto function in many inflammatory processes, such as leukocyte migration, as well as angiogenesis, cancer growth, and metastasis. The interactions between %CCL25% and CCR9 have been implicated in leukocyte trafficking to the small %intestine%, a frequent metastatic site for ovarian cancer cells. A common feature ofovarian tumor microenvironment is hypoxia and under these experimental conditions we compared the chemokine receptor profiles expressed by normal ovarian epithelial cells as well as ovarian cell lines (NIH:OVCAR-3, TOV-112D, Caov-3 and SKOV-3). We report that CCR9 expression is significantly elevated in ovarian cancer cell lines, when compared to normal ovarian epithelial cells. The migration and invasion potential of ovarian cancer cells were impaired following neutralization of %CCL25%/CCR9 interactions using anti-CCR9 and anti-CCR9 antibodies. %CCL25% also modulated the expression of the collagenases, gelatinases, and stromalysins in ovarian cancer cells. These studies indicate that interactions between %CCL25% and CCR9 may contribute to ovarian cancer metastasis.

2/7/60 (Item 16 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18118740 BIOSIS NO.: 200500025805
Interactions of mucosal lymphocytes with the extracellular matrix
AUTHOR: Dotan Iris; Vigodman Sharon; Broitman Lena; Tulchinsky Hagit; Abram Talia; Zhanin-Zhorov Alexandra; Hecht Iris; Gur Hanan; Halpern Zamir; Lider Ofer
JOURNAL: JPGN Journal of Pediatric Gastroenterology and Nutrition 39 (Suppl. 3): pS774 2004 2004
MEDIUM: print
CONFERENCE/MEETING: 1st ESPGHAN Capri Meeting Naples, Italy May 27-29, 2004; 20040527
SPONSOR: European Society for Paediatric Gastroenterology Hepatology & Nutrition
ISSN: 0277-2116 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/61 (Item 17 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17967357 BIOSIS NO.: 200400338146
%CCL25% (%TECK%) mediates recruitment of CCR9H IGH gut homing lymphocytes to hepatic endothelium in primary sclerosing cholangitis
AUTHOR: Grant A J (Reprint); Eksteen B; Miles A; Lalor P F; Hubscher S G; Briskin M; Adams D H
AUTHOR ADDRESS: Dept PatholQueen Elizabeth Hosp, Univ Birmingham, Birmingham, W Midlands, B15 2TT, England**England
JOURNAL: Gut 53 (Suppl. 3): pA11-A12 April 2004 2004
MEDIUM: print
CONFERENCE/MEETING: 2004 Annual Meeting of the British Society of Gastroenterology Glasgow, Scotland, UK March 21-24, 2004; 20040321
SPONSOR: British Society of Gastroenterology
ISSN: 0017-5749 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/62 (Item 18 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17745794 BIOSIS NO.: 200400116551
%CCL25% mediates recruitment of CCR9+ gut homing lymphocytes to the liver in primary sclerosing cholangitis.
AUTHOR: Eksteen Bertus (Reprint); Grant Allister J (Reprint); Miles Alice (Reprint); Lalor Patricia F (Reprint); Hubscher Stefan G (Reprint); Briskin Michael; Adams David H (Reprint)
AUTHOR ADDRESS: University of Birmingham, Birmingham, UK**UK
JOURNAL: Hepatology 38 (4 Suppl. 1): p205A October 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 54th Annual Meeting of the American Association for the Study of Liver Diseases Boston, MA, USA October 24-28, 2003; 20031024
SPONSOR: American Association for the Study of Liver Diseases
ISSN: 0270-9139 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: Primary sclerosing cholangitis (PSC) is a chronic inflammatory liver disease characterized by portal inflammation and progressive bile duct destruction. Most patients with PSC give a history of inflammatory %bowel% disease (IBD) leading to the hypothesis that PSC is driven by the inappropriate recruitment of mucosal lymphocytes into the liver. We have previously shown that some lymphocyte homing-receptors are shared by the liver and gut including the endothelial adhesion molecules vascular adhesion protein-1 (VAP-1) and MADCAM-1. However efficient recruitment of lymphocytes also requires an appropriate chemokine-mediated signal. Lymphocyte recruitment to the %bowel% is driven by the restricted expression of the chemokine %CCL25% which activates the recruitment of alpha4beta7+ mucosal lymphocytes bearing its receptor CCR9. Aim: To investigate the role of %CCL25% in mediating the recruitment of gut-associated lymphocytes to the liver. Methods: Liver tissue and paired blood samples were obtained at liver transplantation and small %bowel% from tissue removed during %intestinal% resection. Liver infiltrating lymphocytes (LIL) were isolated by mechanical homogenization and density gradient centrifugation and phenotyped by flow cytometry (FACS). %CCL25% in tissue was demonstrated by dual immuno-fluorescence and confocal microscopy. Migration and adhesion assays were used to confirm CCR9 function. Results: PSC LIL contained a significant population of CCR9high T-cells compared with controls and matched peripheral blood (CCR9+ PSC LIL19% vs. normal liver LIL 1%, p=0.008). CCR9+ lymphocytes co-expressed the gut homing integrin alpha4beta7. %CCL25% was detected on hepatic endothelium in PSC where it co-localised with CD31 but was absent from hepatic endothelium in other liver diseases. In normal liver %CCL25% was restricted to the occasional Kupfer cell where it co-localised with CD11c. PSC LIL migrated to %CCL25% in in-vitro chemotaxis assays and %CCL25% (10ng/ml) was able to trigger a 20 fold increase in adhesion of PSC LIL to immobilised MAdCAM-1 under flow whereas normal LIL showed no response (p=0.002). %CCL25% mediated adhesion to MAdCAM-1 was abolished by pertussis toxin indicating that the effect is mediated via G-protein coupled receptors. Conclusions: 1) Aberrant hepatic %CCL25% recruits CCR9+ mucosal lymphocytes to the liver in PSC by triggering adhesion to MADCAM-1 expressed on hepatic endothelium and is likely to be critical in the maintenance of chronic inflammation in PSC, 2) The presence of high numbers of intrahepatic CCR9+/alpha4beta7+lymphocytes in PSC is evidence that mucosal lymphocytes, are important in the pathogenesis of PSC.

2/7/63 (Item 19 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17622340 BIOSIS NO.: 200300573017
ADHERENCE TO A GLUTEN FREE DIET IS THE MAIN DETERMINANT OF CHEMOKINE EXPRESSION IN CELIAC DISEASE.
AUTHOR: Beck Paul L (Reprint); Li Annie
AUTHOR ADDRESS: Calgary, AB, Canada**Canada
JOURNAL: Digestive Disease Week Abstracts and Itinerary Planner 2003 p Abstract No. W1370 2003 2003
MEDIUM: e-file
CONFERENCE/MEETING: Digestive Disease 2003 FL, Orlando, USA May 17-22, 2003; 20030517
SPONSOR: American Association for the Study of Liver Diseases
American Gastroenterological Association
American Society for Gastrointestinal Endoscopy
Society for Surgery of the Alimentary Tract
DOCUMENT TYPE: Meeting; Meeting Poster; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Celiac disease is a common condition resulting in a reaction to gluten in the diet of those with the disease. Several studies have assessed the cell populations and cytokines involved in the pathogenesis of this disease state. To our knowledge chemokine regulation in celiac disease has never been assessed. Aim; To assess chemokine expression in patients with biopsy proven celiac disease. Methods; 10 consecutive patients with biopsy confirmed celiac disease were studied along with a control population undergoing endoscopy for other indications. All had biopsy confirmed celiac disease at the time of the study. Tissue biopsies were processed in RNA-later and then Trizol. A multi-template RNA

protection assay (RPA) was carried out for all chemokines in this study except for %TECK% (in which RT-PCR was performed). %TECK% is a chemokine, which is specifically expressed in the small %bowel% and has been recently shown to be elevated in patients with small %bowel% %Crohn%'s disease. Results; 4 patients had been on a gluten free diet for a min. of 6 months. The others were newly diagnosed cases. Compared to controls, celiac patients had marked elevated Ltn (p=0.03), IP-10 (p=0.02), MIP-1b (p=0.02), MIP-1a (p=0.02), MCP-1 (p=0.01), IL-8 (p=0.03) and I-309 (p=0.02), %TECK% (p= 0.003). When the celiac patients were assessed as those on a gluten free diet and untreated, all of the above chemokines were significantly (p>0.05) higher in the untreated vs diet treated patients. When celiac patients were divided, via pathology, into mild vs moderate to severe independent of diet none of chemokines were significantly different suggesting that adherence to a gluten free diet is a more important determinant of chemokine expression than pathology findings. Further study of chemokine expression may add to our understanding of the pathogenesis of celiac disease..

2/7/64 (Item 20 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17621124 BIOSIS NO.: 200300571801
IN VIVO EVIDENCE FOR FUNCTIONAL ROLE OF %TECK% IN T LYMPHOCYTE-ENDOTHELIUM INTERACTION IN INFLAMED AND UNINFLAMED %INTESTINAL% MUCOSA.
AUTHOR: Hosoe Naoki (Reprint); Miura Soichiro; Watanabe Chikako; Tsuzuki Yoshikazu; Hokari Ryota; Teramoto Ken; Ogawa Toshiko; Inamura Toshiaki; Oyama Tokushige; Fujiyama Yoichi; Nagata Hiroshi; Ishii Hiromasa
AUTHOR ADDRESS: Tokyo, Japan**Japan
JOURNAL: Digestive Disease Week Abstracts and Itinerary Planner 2003 p Abstract No. S1098 2003 2003
MEDIUM: e-file
CONFERENCE/MEETING: Digestive Disease 2003 FL, Orlando, USA May 17-22, 2003; 20030517
SPONSOR: American Association for the Study of Liver Diseases
American Gastroenterological Association
American Society for Gastrointestinal Endoscopy
Society for Surgery of the Alimentary Tract
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: The recirculation of lymphocytes through %intestinal% mucosa is important for specific immune defense. Recently role of C-C chemokines and their receptors in organ-specific homing of lymphocytes has been suggested, however, there are not enough in vivo evidences for that. The aim of this study was to examine whether %TECK% and its receptor CCR 9 are involved in the dynamic process of T-lymphocytes-endothelium interaction in microvessels of murine small and large %intestinal% mucosa by an intravital microscope at uninflamed as well as TNF-alpha induced inflammatory conditions. Methods: T lymphocytes were isolated from small %intestinal% lamina propria (LPL) and %intestinal% epithelium (IEL). Mucosal lymphocytes were labeled with fluorochrome carboxyfluorescein diacetate succinimidyl ester (CFSE) and administered from jugular vein of the recipient mice. Adhesion of fluorescence labeled lymphocytes to microvessels of small %intestinal% and colonic mucosa was observed under an intravital fluorescence microscope. In some experiments, TNF-alpha (25 epsilong/g) was injected intraperitoneally to mice 5 hours before lymphocyte injection. In another sets of experiments, LPLs and IELs were desensitized with excess amount of %TECK% and changes in cell kinetics was examined. Expression of %TECK% mRNA was examined by PCR and distribution of %TECK% protein was examined by immunohistochemistry and by in situ demonstration with fluorescein-labeled anti-%TECK%. Results: Intravital observation demonstrated that LPLs and IELs were time-dependently accumulated in the mucosal microvessels of small %intestine% and %colon%. Desensitization of CCR9 with %TECK% significantly inhibited the LPL and IEL migration toward the small %intestine%, but not in the %colon%. TNF-alpha induced a significant increase in LPL migration at the small %intestine% and %colon%, while TNF-alpha increased IEL only in the small %intestine%. Desensitization of CCR9 with %TECK% also significantly attenuated the TNF-alpha induced LPL and IEL migration in the small %intestine%. Increased expression of %TECK% induced by TNF-alpha was observed in the lamina propria and %intestinal% epithelial cells of small %intestine% . Conclusions: It was demonstrated in situ that %TECK% plays an important role in LPLs and IEL adherence to the microvessels of small %intestine%, but not %colon%, under uniflamed as well as inflamed conditions..

2/7/65 (Item 21 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17397732 BIOSIS NO.: 200300356451
Constitutive Expression of the Chemokine Receptor CCR9 in Bone Marrow Enhanced Thymocyte Maturation and Mucosal Trafficking.
AUTHOR: Straley Erin E (Reprint); Kelleher Erin M (Reprint); Georgantas Robert W (Reprint); Calabresi Peter A (Reprint); Pardoll Drew M (Reprint) ; Powell Jonathan D (Reprint); Whartenby Katharine A (Reprint); Civin Curt I (Reprint)
AUTHOR ADDRESS: Immunology and Hematopoiesis, Sidney Kimmel Cancer Center at Johns Hopkins, Baltimore, MD, USA**USA
JOURNAL: Blood 100 (11): pAbstract No. 445 November 16, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: 44th Annual Meeting of the American Society of Hematology Philadelphia, PA, USA December 06-10, 2002; 20021206
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Poster; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Chemokine receptors are known to be important homing molecules but their role in cell function has been less well defined. CCR9 has been implicated in homing of pre-T cells to the thymus, although other molecules may also mediate this process. The ligand for CCR9, thymus expressed chemokine (%TECK%/%CCL25%) is expressed only in the thymus and %intestinal% compartments and is thought to have a role in thymocyte trafficking within the thymus. We undertook studies to assess whether constitutive expression of CCR9 in bone marrow transplant would affect homing of bone marrow progenitors to the thymus and gut lymphoid tissue. Thymic reconstitution was measured after a myeloablative syngeneic bone marrow transplant in which CCR9 was overexpressed in murine bone marrow stem-progenitor cells by lentiviral-mediated transduction. While homing of the progenitors to the thymus did not appear to be significantly affected, maturation of thymocytes was increased, leading to an increased number of CD4 or CD8 single positive thymocytes as shown in the figure below. Constitutive expression of CCR9 did not prevent emigration of cells from the thymus, as CCR9-expressing T cells were observed in Peyer's patches. Additional studies showed that modification of cells with CCR9 led to inhibition of apoptosis. In thymocytes, binding of ligand did not lead to an upregulation of bcl-2 or bcl-x but did enhance activation of Erk 1/2, suggesting that it activates signaling pathways in thymocytes. Thus, CCR9 may play a role in development of thymocytes by enhancing the double positivefwdarw single positive transition, possibly through inhibition of apoptosis.

2/7/66 (Item 22 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17156957 BIOSIS NO.: 200300115676
%Gastrointestinal% intraepithelial lymphocytes and T cell lymphomas.
AUTHOR: Farstad I N (Reprint); Lundin K E A
AUTHOR ADDRESS: Institute of Pathology, Rikshospitalet, N-0027, Oslo, Norway**Norway
AUTHOR E-MAIL ADDRESS: i.n.farstad@labmed.uio.no
JOURNAL: Gut 52 (2): p163-164 February 2003 2003

MEDIUM: print
ISSN: 0017-5749 _(ISSN print)
DOCUMENT TYPE: Article; Editorial
RECORD TYPE: Citation
LANGUAGE: English

2/7/67 (Item 23 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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16937668 BIOSIS NO.: 200200531179
Interferon-gamma induces the expression of the chemokine %TECK% in Caco-2 but not HT-29 %intestinal% epithelial cells
AUTHOR: Papadakis Konstantinos A (Reprint); Moreno Sofia T (Reprint); Targan Stephan R (Reprint)
AUTHOR ADDRESS: Los Angeles, CA, USA**USA
JOURNAL: Gastroenterology 122 (4 Suppl. 1): pA.530 April, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: Digestive Disease Week and the 103rd Annual Meeting of the American Gastroenterological Association San Francisco, CA, USA May 19-22, 2002; 20020519
ISSN: 0016-5085
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/68 (Item 24 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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16936990 BIOSIS NO.: 200200530501
In situ demonstration of role of C-C chemokine receptors 6 and 9 in T lymphocyte migration to the inflamed small %intestine% and %colon%
AUTHOR: Hosoe Naoki (Reprint); Watanabe Chikako; Miura Soichiro; Tsuzuki Yoshikazu; Hokari Ryota; Shigematsu Takeharu; Teramoto Ken; Okada Yoshikiyo; Nagata Hiroshi; Ishii Hiromasa
AUTHOR ADDRESS: Tokyo, Japan**Japan
JOURNAL: Gastroenterology 122 (4 Suppl. 1): pA-399 April, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: Digestive Disease Week and the 103rd Annual Meeting of the American Gastroenterological Association San Francisco, CA, USA May 19-22, 2002; 20020519
ISSN: 0016-5085
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/69 (Item 25 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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16925213 BIOSIS NO.: 200200518724
Cholera Toxin enhances the expression of adhesion molecules and B lymphocyte migration in %intestinal% mucosa
AUTHOR: Okada Yoshikiyo (Reprint); Tsuzuki Yoshikazu (Reprint); Hokari Ryota (Reprint); Miyazaki Junichi (Reprint); Matsuzaki Koji (Reprint); Kawaguchi Atsushi (Reprint); Nagao Shigeaki (Reprint); Iwai Atsuhiro (Reprint); Miyahara Tohru (Reprint); Itoh Kazuro (Reprint); Miura Soichiro (Reprint)
AUTHOR ADDRESS: Tokorozawa, Japan**Japan
JOURNAL: Gastroenterology 122 (4 Suppl. 1): pA-152-A-153 April, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: Digestive Disease Week and the 103rd Annual Meeting of the American Gastroenterological Association San Francisco, CA, USA May 19-22, 2002; 20020519
ISSN: 0016-5085
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/70 (Item 26 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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16849494 BIOSIS NO.: 200200443005
Analysis of leukocyte population changes in lactoferrin-fed mice
AUTHOR: Wakabayashi H (Reprint); Takakura N (Reprint); Kuwata H (Reprint); Yamauchi K (Reprint); Teraguchi S (Reprint); Hayasawa H (Reprint)
AUTHOR ADDRESS: Nutritional Science Laboratory, Morinaga Milk Industry Co. Ltd., Zama, Kanagawa, 228-8583, Japan**Japan
JOURNAL: Biochemistry and Cell Biology 80 (1): p145 2002 2002
MEDIUM: print
CONFERENCE/MEETING: 5th International Conference on Lactoferrin: Structure, Function and Applications Banff, Alberta, Canada May 04-09, 2001; 20010504
ISSN: 0829-8211
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/71 (Item 27 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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15870183 BIOSIS NO.: 200100042022
Expression of thymus expressed chemokine (%TECK%) and its receptor CCR9 in the small %bowel%: Enhanced recirculation of CCR9+lymphocytes in small %bowel% %Crohn%'s disease
AUTHOR: Papadakis K A (Reprint); Prehn J (Reprint); Andrew D P; Ponath P D; Targan S R (Reprint)
AUTHOR ADDRESS: IBD Center, Cedars-Sinai Medical Center, Los Angeles, CA, 90048, USA**USA
JOURNAL: FASEB Journal 14 (6): pA975 April 20, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: Joint Annual Meeting of the American Association of Immunologists and the Clinical Immunology Society Seattle, Washington, USA May 12-16, 2000; 20000512
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/72 (Item 28 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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15841494 BIOSIS NO.: 200100013333
Regional expression of thymus expressed chemokine (%TECK%) and its receptor CCR9 in the small %bowel%: Evidence for enhanced recirculation of CCR9+ lymphocytes in small %bowel% %Crohn%'s disease
AUTHOR: Papadakis K A (Reprint); Prehn J (Reprint); Andrew D P (Reprint); Ponath P D; Targan S R
AUTHOR ADDRESS: Inflammatory Bowel Disease Center, Cedars-Sinai Medical Center, UCLA School of Medicine, Los Angeles, CA, USA**USA
JOURNAL: Digestive and Liver Disease 32 (Supplement 1): pA44 May, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: International Meeting on Inflammatory Bowel Diseases Capri, Italy June 18-21, 2000; 20000618
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/73 (Item 29 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15797066 BIOSIS NO.: 200000515379
Selective expression of %TECK%/CCR9 in the small %intestine% suggests a unique role for this chemokine receptor pair in small %intestinal% immunity
AUTHOR: Agace W W (Reprint)
AUTHOR ADDRESS: Immunology Unit, Department of Cell and Molecular Biology, Lund University, Lund, Sweden**Sweden
JOURNAL: Scandinavian Journal of Immunology 52 (4): p455 October, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 2nd European Mucosal Immunity Group Meeting Gothenburg, Sweden October 06-08, 2000; 20001006
ISSN: 0300-9475
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/74 (Item 30 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15795672 BIOSIS NO.: 200000513985
GPR-9-6/CCR9 is selectively expressed on %intestinal% homing lymphocytes and thymocytes and is necessary for %TECK% mediated chemotaxis
AUTHOR: Andrew D P (Reprint); Campbell J J; Heath H M (Reprint); Kassam N (Reprint); Qin S (Reprint); Soler D (Reprint); Rottman J B (Reprint); Butcher E C; Ponath P D (Reprint); Agace W W; Zabel B A (Reprint)
AUTHOR ADDRESS: Millennium Pharmaceuticals, Cambridge, MA, 02142, USA**USA
JOURNAL: Tissue Antigens 55 (Supplement 1): p44 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 7th Workshop and Conference on Human Leucocyte Differentiation Antigens Harrogate, England, UK June 20-24, 2000; 20000620
ISSN: 0001-2815
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/75 (Item 31 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13809664 BIOSIS NO.: 199799443724
%TECK%: A novel CC chemokine associated with T-cell development
AUTHOR: Vicari A P; Figueroa D; Zlotnik A
AUTHOR ADDRESS: DNAX Res. Inst., Palo Alto, CA, USA**USA
JOURNAL: Journal of Allergy and Clinical Immunology 99 (1 PART 2): pS246 1997 1997
CONFERENCE/MEETING: Joint Meeting of the American Academy of Allergy, Asthma and Immunology, the American Association of Immunologists and the Clinical Immunology Society San Francisco, California, USA February 21-26, 1997; 19970221
ISSN: 0091-6749
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/76 (Item 1 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

03344898 2006130614
Redundant role of chemokines %CCL25%/%TECK% and CCL28/MEC in IgASUP+ plasmablast recruitment to the %intestinal% lamina propria after rotavirus infection
Feng N.; Jaimes M.C.; Lazarus N.H.; Monak D.; Zhang C.; Butcher E.C.; Greenberg H.B.
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NO. OF REFERENCES: 43

Rotaviruses (RV) are the most important cause of severe childhood diarrheal disease. In suckling mice, infection with RV results in an increase in total and virus-specific IgASUP+ plasmablasts in the small %intestinal% lamina propria (LP) soon after infection, providing a unique opportunity to study the mechanism of IgASUP+ cell recruitment into the small %intestine%. In this study, we show that the increase in total and RV-specific IgASUP+ plasmablasts in the LP after RV infection can be blocked by the combined administration of Abs against chemokines %CCL25% and CCL28, but not by the administration of either Ab alone. RV infection in CCR9 knockout mice still induced a significant accumulation of IgASUP+ plasmablasts in the LP, which was blocked by the addition of anti-CCL28 Ab, confirming the synergistic role of %CCL25% and CCL28. The absence of IgASUP+ plasmablast accumulation in LP following combined anti-chemokine treatment was not due to changes in proliferation or apoptosis in these cells. We also found that coadministration of anti-%CCL25% and anti-CCL28 Abs with the addition of anti-alphaSUB4 Ab did not further inhibit IgASUP+ cell accumulation in the LP and that the %CCL25% receptor, CCR9, was coexpressed with the %intestinal% homing receptor alphaSUB4betaSUB7 on IgASUP+ plasmablasts. Finally, we showed that RV infection was associated with an increase in both %CCL25% and CCL28 in the small %intestine%. Hence, our findings indicate that alphaSUB4betaSUB7 along with either CCR9 or CCR10 are sufficient for mediating the %intestinal% migration of IgASUP+ plasmablasts during RV infection. Copyright (c) 2006 by The American Association of Immunologists, Inc.

2/7/77 (Item 2 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

03325190 2006106116
CD8SUP+ recent thymic emigrants home to and efficiently repopulate the small %intestine% epithelium
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ISSN: 1529-2908 eISSN: 1529-2916
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Prevailing knowledge dictates that naive alphabeta T cells require activation in lymphoid tissues before differentiating into effector or memory T cells capable of trafficking to nonlymphoid tissues. Here we demonstrate that CD8SUP+ recent thymic emigrants (RTEs) migrated directly into the small %intestine%. CCR9, %CCL25% and alphaSUB4 betaSUB7 integrin were required for gut entry of CD8SUP+ RTEs. After T cell receptor stimulation, %intestinal% CD8SUP+ RTEs proliferated and acquired a surface phenotype resembling that of intraepithelial lymphocytes. CD8SUP+ RTEs efficiently populated the gut of lymphotoxin-alpha-deficient mice, which

lack lymphoid organs. These studies challenge the present understanding of naive alphabeta T cell trafficking and suggest that RTEs may be involved in maintaining a diverse immune repertoire at mucosal surfaces. (c) 2006 Nature Publishing Group.

2/7/78 (Item 3 from file: 71)
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02843238 2004320179
Hepatic endothelial %CCL25% mediates the recruitment of CCR9SUP+ gut-homing lymphocytes to the liver in primary sclerosing cholangitis
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Journal: Journal of Experimental Medicine, 200/11 (1511-1517), 2004, United States
CODEN: JEMEA
ISSN: 0022-1007
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 26

Primary sclerosing cholangitis (PSC), a chronic inflammatory liver disease characterized by progressive bile duct destruction, develops as an extra-%intestinal% complication of inflammatory %bowel% disease (IBD) (Chapman, R.W. 1991. Gut. 32:1433-1435). However, the liver and %bowel% inflammation are rarely concomitant, and PSC can develop in patients whose colons have been removed previously. We hypothesized that PSC is mediated by long-lived memory T cells originally activated in the gut, but able to mediate extra-%intestinal% inflammation in the absence of active IBD (Grant, A.J., P.F. Lalor, M. Salmi, S. Jalkanen, and D.H. Adams. 2002. Lancet. 359:150-157). In support of this, we show that liver. infiltrating lymphocytes in PSC include mucosal T cells recruited to the liver by aberrant expression of the gut-specific chemokine %CCL25% that activates alpha4beta7 binding to mucosal addressin cell adhesion molecule 1 on the hepatic endothelium. This is the first demonstration in humans that T cells activated in the gut can be recruited to an extra-%intestinal% site of disease and provides a paradigm to explain the pathogenesis of extra-%intestinal% complications of IBD.

2/7/79 (Item 4 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE
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02741857 2004217605
CCR2 expressing CD4SUP+ T lymphocytes are preferentially recruited to the ileum in %Crohn%'s disease
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Journal: Gut, 53/9 (1287-1294), 2004, United Kingdom
CODEN: GUTTA
ISSN: 0017-5749
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 51

Background and aims: Chemokine receptors are key determinants of leucocyte trafficking. While the chemokine receptor CCR9 and its chemokine ligand %CCL25% (%TECK%) mediate lymphocyte homing to the healthy small %intestine% , the chemokine receptors important for recruitment during %intestinal% inflammation are undefined. Animal studies have suggested potential roles for CCR2 and CCR5 in inflammatory %bowel% disease (IBD). The aim of this study was to understand the role of CCR2 in human IBD. Methods: Resections of ileum or %colon% were obtained from patients undergoing surgery for small %bowel% %Crohn%'s disease (SBCD; n = 10), %Crohn%'s colitis (n = 5), ulcerative colitis (n = 6), and non-IBD related conditions (control ileum n = 11; control %colon% n = 11). Expression of CCR2 by lamina propria lymphocytes (LPLs) was determined by both flow cytometry and immunohistochemistry. As a functional correlate, chemotaxis assays using the CCR2 ligand, CCL2 (MCP-1), were performed. Expression of CCR2 by peripheral blood lymphocytes was determined by flow cytometry. Results: There were greater than 30-fold more CCR2SUP+ LPLs in SBCD than in control ileum (29.3% (19.9-55.1) v 0.9% (0.4-11.5); p = 0.0007). Specifically, CCR2 SUP+CD4SUP+ LPLs were increased (p = 0.002) whereas CCR2 SUP+CD8SUP+ LPLs were not. Increased expression included both memory (CD45ROSUP+ p = 0.005) and naive (CD45ROSUP- p = 0.01) CCR2SUP+ populations. The increase in CCR2SUP+ LPLs in SBCD was confirmed by both immunohistochemistry (p = 0.0002) and enhanced chemotactic responses to CCL2. CCR2 expression was not increased in the peripheral blood of patients with SBCD, suggesting ongoing recruitment of the CCR2SUP+ population to the ileum. In contrast with SBCD, there was no significant increase in CCR2SUP+ LPLs in %Crohn%'s colitis or ulcerative colitis samples. Conclusions: The chemokine receptor CCR2 appears to be an important contributor to accumulation of CD4SUP+ T lymphocytes in the ileum in small %bowel% %Crohn%'s disease. Blockade of CCR2 may provide a novel therapeutic alternative.

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02222594 2003006308
Involvement of %CCL25% (%TECK%) in the generation of the murine small-%intestinal% CD8alphaalphaSUP+CD3SUP+ intraepithelial lymphocyte compartment
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Journal: European Journal of Immunology, 32/12 (3488-3497), 2002, Germany
PUBLICATION DATE: December 1, 2002
CODEN: EJIMA
ISSN: 0014-2980
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 39

The CC chemokine %CCL25% (%TECK%) is selectively expressed in the thymus and small %intestine%, indicating a potential role in T lymphocyte development. In the present study we examined the role of %CCL25% in the generation of the small-%intestinal% CD8alphaalphaSUP+CD3SUP+ intraepithelial lymphocyte (IEL) compartment. %CCL25% mRNA expression in the murine small %intestine% increased at three weeks of age and corresponded with the appearance of CD8alphaalphaSUP+CD3SUP+ lymphocytes in the small-%intestinal% epithelium. Administration of monoclonal neutralizing anti-%CCL25% antibody to two-week-old mice led to a (similar)50% reduction in the total number of CD8alphaalphaSUP+TCRgammadeltaSUP+ and CD8alphaalphaSUP+TCRalphabetaSUP+ IEL at four weeks of age. Freshly isolated murine CD8alphaalphaSUP+CD3SUP+ IEL migrated in response to %CCL25% and expressed the %CCL25% receptor, CCR9. Analysis of CCR9 expression on putative IEL precursor populations demonstrated the presence of both CCR9SUP- and CCR9SUP+ cells and indicated that up-regulation of this receptor occurred during IEL precursor differentiation. Finally, data from wild-type and RAGSUP-/- mice suggested that the reduction in CD8alphaalphaSUP+CD3SUP+ IEL in anti-%CCL25% antibody treated mice resulted primarily from defective maintenance and/or development of IEL precursors rather than a direct effect on mature CD8alphaalphaSUP+CD3SUP+ IEL.

2/7/81 (Item 6 from file: 71)

DIALOG(R)File 71:ELSEVIER BIOBASE

02163306 2002243443
%CCL25% mediates the localization of recently activated CD8alphabetaSUP+ lymphocytes to the small-%intestinal% mucosa
Svensson M.; Marsal J.; Ericsson A.; Carramolino L.; Broden T.; Marquez G.; Agace W.W.
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Journal: Journal of Clinical Investigation, 110/8 (1113-1121), 2002, United States
CODEN: JCINA
ISSN: 0021-9738
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 29

The recruitment of antigen-specific T lymphocytes to the %intestinal% mucosa is central to the development of an effective mucosal immune response, yet the mechanism by which this process occurs remains to be fully defined. Here we show that the CC chemokine receptor 9 (CCR9) is selectively and functionally expressed on murine alphaSUBEbetaSUB7SUP+ naive CD8alphabetaSUP+ lymphocytes and a subset of recently activated CD69SUP+ CD8alphabetaSUP+ lymphocytes. Using a T cell receptor transgenic transfer model, we demonstrate that CCR9 expression is functionally maintained on CD8alphabetaSUP+ lymphocytes following activation in mesenteric lymph nodes but rapidly downregulated on CD8alphabetaSUP+ lymphocytes activated in peripheral lymph nodes. These recently activated CCR9SUP+ CD8alphabetaSUP+ lymphocytes selectively localized to the small-%intestinal% mucosa, and in vivo neutralization of the CCR9 ligand, %CCL25% , reduced the ability of these cells to populate the small-%intestinal% epithelium. Together these results demonstrate an important role for chemokines in the localization of T lymphocytes to the small-%intestinal% mucosa and suggest that targeting %CCL25% and/or CCR9 may provide a means to selectively modulate small-%intestinal% immune responses.

2/7/82 (Item 7 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

02147355 2002227656
Mice lacking the CCR9 CC-chemokine receptor show a mild impairment of early T- and B-cell development and a reduction in T-cell receptor gammadeltaSUP+ gut intraepithelial lymphocytes
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Journal: Blood, 98/9 (2626-2632), 2001, United States
PUBLICATION DATE: November 1, 2001
CODEN: BLOOA
ISSN: 0006-4971
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 29

CC chemokine receptor (CCR) 9, the receptor for the CC-chemokine %CCL25% /thymus-expressed chemokine (%TECK%), is mainly expressed by thymocytes and by intraepithelial (IEL) and lamina propria lymphocytes of the small %intestine%. To study the biologic role of CCR9, a mouse strain was generated in which the CCR9 gene was deleted. In spite of the high level of CCR9 found in double- and single-positive thymocytes and of the expression of its corresponding ligand on thymic stromal cells, CCR9 deletion had no major effect on intrathymic T-cell development. It was noted that there was only a one-day lag in the appearance of double-positive cells during fetal ontogeny in CCR9SUP-/- thymi. When tested in chemotaxis assay, thymocytes isolated from CCR9SUP-/- mice failed to respond to %TECK%/%CCL25%. Taken together, these results suggest that in thymocytes, CCR9 is the only physiologic receptor for %TECK%/%CCL25%, and that it is dispensable for proper T-cell development. Bone marrow pre-pro-B cells migrate in response to %TECK%/%CCL25%, but more mature B cells do not. Consistent with this observation, it was shown that there are fewer pre-pro-B cells in CCR9SUP-/- mice than in wild-type mice. However, this diminution does not appear to have a detectable effect on the generation of a normal complement of mature B cells. Finally, it was shown that in the small %intestine% of CCR9-deficient mice, the intraepithelial T-cell-to-epithelial cell ratio is decreased, an observation that can be accounted for by a marked diminution of the T-cell receptor gammadeltaSUP+ compartment. (c) 2001 by The American Society of Hematology.

2/7/83 (Item 8 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

01940293 2002020622
Rapid acquisition of tissue-specific homing phenotypes by CD4SUP+ T cells activated in cutaneous or mucosal lymphoid tissues
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Journal: Journal of Experimental Medicine, 195/1 (135-141), 2002, United States
PUBLICATION DATE: January 7, 2002
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DOCUMENT TYPE: Article
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NO. OF REFERENCES: 27

Effector and memory T cells can be subdivided based on their ability to traffic through peripheral tissues such as inflamed skin and %intestinal% lamina propria, a property, controlled by expression of 'tissue-specific' adhesion and chemoattractant receptors. However, little is known about the development of these selectively homing T cell subsets, and it is unclear whether activation in cutaneous versus %intestinal% lymphoid organs directly results in effector/memory T cells that differentially express adhesion and chemoattracant receptors targeting them to the corresponding nonlymphoid site. We define two murine CD4SUP+ effector/memory T cell subsets that preferentially localize in cutaneous or %intestinal% lymphoid organs by their reciprocal expression of the adhesion molecules P-selectin ligand (P-lig) and alpha4beta7, respectively. We show that within 2 d of systemic immunization CD4SUP+ T cells activated in cutaneous lymph nodes upregulate P-lig, and downregulate alpha4beta7, while those responding to antigen in %intestinal% lymph nodes selectively express high levels of alpha4beta7 and acquire responsiveness to the %intestinal% chemokine thymus-expressed chemokine (%TECK%). Thus, during an immune response, local microenvironments within cutaneous and %intestinal% secondary, lymphoid organs differentially direct T cell expression of these adhesion and chemoattractant receptors, targeting the resulting effector T cells to the inflamed skin or %intestinal% lamina propria.

2/7/84 (Item 9 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

01922934 2002003877
Characterization of CCR9 expression and %CCL25%/thymus-expressed chemokine responsiveness during T cell development: CD3SUPhighCD69SUP+ thymocytes and gammadeltaTCRSUP+ thymocytes preferentially respond to %CCL25%
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Journal: Journal of Immunology, 168/1 (134-142), 2002, United States
PUBLICATION DATE: January 1, 2002
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NO. OF REFERENCES: 32

CCR9 mediates chemotaxis of thymocytes in response to %CCL25% /thymus-expressed chemokine, and its mRNA is selectively expressed in thymus and small %intestine%, the two known sites of T lymphopoiesis. To examine the expression of CCR9 during lymphocyte development, we generated polyclonal Ab that recognizes murine CCR9. CCR9 was expressed on the majority of immature CD4SUP+CD8SUP+ (double-positive) thymocytes, but not on immature CD4SUP-CD8SUP- (double-negative) thymocytes. CCR9 was down-regulated during the transition of double-positive thymocytes to the CD4SUP+ or CD8SUP+ (single- positive) stage, and only a minor subset of CD8SUP+ lymph node T cells expressed CCR9. All CCR9SUP+ thymocyte subsets migrated in response to %CCL25%; however, CD69SUP+ thymocytes demonstrated enhanced %CCL25%-induced migration compared with CD69SUP- thymocytes. Ab-mediated TCR stimulation also enhanced %CCL25% responsiveness, indicating that %CCL25%-induced thymocyte migration is augmented by TCR signaling. Approximately one-half of all gammadeltaTCRSUP+ thymocytes and peripheral gammadeltaTCRSUP+ T cells expressed CCR9 on their surface, and these cells migrated in response to %CCL25%. These findings suggest that CCR9 may play an important role in the development and trafficking of both alphabetaTCRSUP+ and gammadeltaTCRSUP+ T cells.

2/7/85 (Item 10 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE
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01879392 2001241322
Age-related changes in CCR9SUP+ circulating lymphocytes: Are CCR9SUP+ naive T cells recent thymic emigrants?
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Journal: Scandinavian Journal of Immunology, 54/5 (435-439), 2001, United Kingdom
CODEN: SJIMA
ISSN: 0300-9475
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 20

The chemokine receptor CCR9 is reported to be predominantly expressed by thymocytes as well as by circulating gut-homing and resident T cells in the small %intestinal% mucosa. Its ligand thymus-expressed chemokine (%TECK%) is produced by thymic and small %intestinal% epithelium. Here we report that the proportion of circulating CCR9SUP+ naive T cells (mostly CD4SUP+) declines with age, from approximately 15% of all T cells at birth to around 1% in adults. The proportion of CCR9SUP+ T cells lacking the classical gut-homing receptor alpha4beta7, was much higher in children than in adults. Therefore, circulating CD3SUP+CCR9SUP+CD45RASUP+ cells have most likely left the thymus quite recently. This notion was supported by the small number of CCR9SUP+ naive T cells which was present shortly after thymectomy. Establishing a phenotypic marker for recent thymic emigrants might provide a powerful tool in the clinical assessment and follow-up after cancer chemotherapy, hematopoietic stem cell transplantation, and during antiretroviral treatment of human immunodeficiency virus (HIV)-infected patients.

2/7/86 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
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13909610 EMBASE No: 2006312627
Chemokines involved in protection from colitis by CD4SUP+CD25 SUP+ regulatory T cells
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Inflammatory Bowel Diseases (INFLAMMATORY BOWEL DIS.) (United States) 2006, 12/7 (612-618)
CODEN: IBDNB ISSN: 1078-0998 eISSN: 1536-4844
PUBLISHER ITEM IDENTIFIER: 00054725200607000000011
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 35

Chemokines are small proteins involved in the direction of migration of immune cells both during normal homeostasis and inflammation. Chemokines have been implicated in the pathology of many different inflammatory disorders and are therefore appealing therapeutic targets. Using a chemokine/chemokine receptor-specific gene expression profiling system of 67 genes, the authors have determined the expression profile of chemokine and chemokine receptor genes in the rectum of colitic mice and in mice that have been protected fromcolitis by CD4SUP+CD25SUP+ regulatory T cells. In mice protected from colitis, the authors found down regulation of the mRNA expression of the inflammatory chemokine receptors CCR1 and CXCR3 and their ligands CXCL9, CXCL10, CCL5, and CCL7. Also the transcripts for CCR9, %CCL25%, CCL17, and CXCL1 are found down regulated in protected compared with colitic animals. In addition, the authors' results suggest that CCL20 is used by CCR6SUP+ regulatory T cells in the complex process of controlling colitis because transcripts for this chemokine were expressed to a higher level in protected animals. The chemokine pathways identified in the present study may be of importance for the development of new targets for anti-inflammatory treatment strategies in human inflammatory %bowel% disease. Copyright (c) 2006 by Lippincott Williams & Wilkins.

2/7/87 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE
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13105276 EMBASE No: 2005163751
Chemokine-mediated control of T cell traffic in lymphoid and peripheral tissues
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Molecular Immunology (MOL. IMMUNOL.) (United Kingdom) 2005, 42/7 (799-809)
CODEN: IMCHA ISSN: 0161-5890
DOCUMENT TYPE: Journal ; Conference Paper
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 83

Antigen-driven T cell education and subsequent pathogen elimination present particular challenges for the immune system. Pathogens generally enter the body at peripheral sites such as the skin, %gastrointestinal% tract or lung, areas from which naive T cells are largely excluded. Instead, naive T cells constantly recirculate through secondary lymphoid organs, such as lymph nodes and Peyer's patches, in search for antigen brought to these locations by means of afferent lymphatic channels. Here, antigen-loaded dendritic cells present antigen-peptide-MHC complexes to clonotypic T cells and provide appropriate co-stimulatory signals for immune response initiation. As a result, short-lived effector T cells and long-lived memory T cells are generated that reach the peripheral tissue for participation in immune responses and immune surveillance. Effector and memory T cell relocation is non-random, due to tissue-specific "address codes" that allow proper tissue homing. This process involves adhesion

molecules, including selectins, integrins, and corresponding vascular ligands as well as the large family of chemokines and their receptors. Here, we discuss the changes in chemokine receptor expression that occur during T cell activation and differentiation, and the ways in which these changes impact on the migration potential of naive, effector, and memory T cells. We summarize our current understanding of T cell homing to the T zone and B cell follicles within secondary lymphoid tissues and highlight the two chemokine receptors CCR7 and CXCR5 that recognize chemokines constitutively present either in the T zone (CCR7 ligands CCL19/ELC and CCL21/SLC) or follicular compartment (CXCR5 ligand CXCL13/BCA-1). CCR7 is characteristic for naive and central memory T (TSUBCM) cells whereas CXCR5 distinguishes follicular B helper T (TSUBFH) cells. In addition, we further subdivide long-lived memory T cells into CCR7-negative effector memory T (TSUBEM) cells and peripheral immune surveillance T (TSUBPS) cells. The latter term designates the extraordinarily large subset of memory T cells with primary residence in normal (healthy) peripheral tissues. Our current understanding of TSUBPS cell migration and function is highly fragmentary, but these cells are thought to provide immediate protection locally at the site of pathogen entry. Here, we propose that the tissue distribution of TSUBPS cells is determined by a distinct set of chemokines and corresponding receptors that differs from those operating in secondary lymphoid tissues and inflammatory sites.

2/7/88 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

12579037 EMBASE No: 2004164508
Chemokine receptors in melanoma: CCR9 has a potential role in metastasis to the small %bowel%
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Journal of Investigative Dermatology (J. INVEST. DERMATOL.) (United States) 2004, 122/3 (xiv-xv)
CODEN: JIDEA ISSN: 0022-202X
DOCUMENT TYPE: Journal ; Editorial
LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 13

2/7/89 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

12557325 EMBASE No: 2004140718
Possible link between unique chemokine and homing receptor expression at diagnosis and relapse location in a patient with childhood T-ALL
Annels N.E.; Willemze A.J.; Van Der Velden V.H.J.; Faaij C.M.J.M.; Van Wering E.; Sie-Go D.M.D.S.; Egeler R.M.; Van Tol M.J.D.; Revesz T.
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Blood (BLOOD) (United States) 01 APR 2004, 103/7 (2806-2808)
CODEN: BLOOA ISSN: 0006-4971
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 15

Childhood acute lymphoblastic leukemia (ALL) is often associated with extramedullary infiltration by leukemic cells at diagnosis or at relapse. To understand the mechanisms behind the dissemination of T-cell ALL (T-ALL) cells this study investigated the homing receptor expression on the blast cells of 11 pediatric T-ALL patients at diagnosis. One patient revealed a unique profile with high expression of the chemokine receptor CCR9 and the integrin CD103 on the T-ALL cells. Both of these molecules are specifically associated with homing to the gut. This finding was clinically significant as the patient later suffered a relapse that was confined to the gut. Immunohistochemistry revealed that the leukemic cells in the gut still expressed CCR9 and colocalized with a high expression of the CCR9 ligand, %CCL25%. These findings suggest that the original expression of CCR9 and CD103 on the leukemic cells contributed to the relapse location in the gut of this patient.

2/7/90 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

11841380 EMBASE No: 2002416219
Homeostatic chemokines and the targeting of regional immunity
Kunkel E.J.; Butcher E.C.
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Advances in Experimental Medicine and Biology (ADV. EXP. MED. BIOL.) (United States) 2002, 512/- (65-72)
CODEN: AEMBA ISSN: 0065-2598
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 28

Tissue-selective trafficking of memory and effector T and B lymphocytes is mediated by unique combinations of adhesion molecules and chemokines. The discovery of several related epithelial-expressed chemokines (%TECK%/ %CCL25% in small %intestine%, CTACK/CCL27 in skin, and MEC/CCL28 in diverse mucosal sites) now highlights an important role for epithelial cells in controlling homeostatic lymphocyte trafficking, including the localization of cutaneous and %intestinal% memory T cells, and of IgA plasma cells. Constitutively expressed epithelial chemokines may help determine the character of local immune responses and contribute to the systemic organization of the immune system.

2/7/91 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

00957079 EMBASE No: 1978085401
Retrograde resection of hepatic lobe for extensive carcinoma of the liver
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Medecine et Chirurgie Digestives (MED. CHIR. DIG.) (France) 1977, 6/2 (87-88)
CODEN: MCDGB
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH

The authors applied retrograde resection of hepatic lobe combined with en block resection of %colon%, omentum, peritoneum or diaphragm for 4 cases of primary carcinoma of the liver in right lobe with invasion or adhesion to the surrounding extrahepatic structures. All cases were operated smoothly with this technique. It is believed that this technique is simpler, safer and useful in treating such an extensive carcinoma of the liver.

2/7/92 (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0393660 DBR Accession No.: 2006-07156 PATENT
Identifying modulator of binding of CCX CKR polypeptide to chemokine, by contacting CCX CKR polypeptide and chemokine in presence of test compound, comparing level of binding of chemokine and polypeptide in presence/absence of compound - involving vector-mediated gene transfer and expression in host cell for inflammation, allergy, systemic anaphylaxis, hypersensitivity response, inflammatory %bowel% disease,

psoriasis, autoimmune disease, rheumatoid arthritis, multiple sclerosis, graft rejection, cancer, retinopathy, macular degeneration, infectious disease and immunosuppressive disease therapy
AUTHOR: GOSLING J; DAIRAGHI D J; HANLEY M; MIAO Z; TALBOT D; SCHALL T J
PATENT ASSIGNEE: CHEMOCENTRYX INC 2006
PATENT NUMBER: US 6998239 PATENT DATE: 20060214 WPI ACCESSION NO.: 2006-151961 (200616)
PRIORITY APPLIC. NO.: US 721341 APPLIC. DATE: 20001121
NATIONAL APPLIC. NO.: US 721341 APPLIC. DATE: 20001121
LANGUAGE: English
ABSTRACT: DERWENT ABSTRACT: NOVELTY - Identifying a modulator of binding of CCX CKR polypeptide to chemokine, involves contacting an isolated/recombinant CCX CKR polypeptide having a fully defined 350 amino acid (SEQ ID No. 2) sequence given in the specification, or its fragment/variant, and the chemokine in presence of a test compound; and comparing the level of binding of chemokine and polypeptide in contacting step with the level of binding in the absence of the test compound. DETAILED DESCRIPTION - Identifying (M1) a modulator of the binding of CCX CKR polypeptide to a chemokine, involves contacting an isolated or recombinant CCX CKR polypeptide having a fully defined 350 amino acid (SEQ ID No. 2) sequence given in the specification, or its fragment or variant, and the chemokine in the presence of a test compound; and comparing the level of binding of the chemokine and the polypeptide in the contacting step, with the level of binding in the absence of the test compound, where a decrease in binding indicates that the test compound is an inhibitor of binding and an increase in binding indicates that the test compound is an enhancer of binding, and where the CCX CKR polypeptide, its fragment or variant can bind the chemokine in the absence of test compound and the variant has at least 90% sequence identity to SEQ ID No. 2, and the chemokine is chosen from EBI-1-ligand chemokine (ELC), secondary lymphoid organ chemokine (SLC), thymus expressed chemokine (%TECK%), B-lymphocyte chemoattractant (BLC), cutaneous T cell attracting chemokine (CTACK), murine macrophage inflammatory protein 1 gamma (mMIP-1gamma) and viral macrophage inflammatory protein II (vMIPII). WIDER DISCLOSURE - The following are disclosed: (1) an isolated or recombinant CCX CKR polypeptide or its immunogenic fragment; (2) an isolated polynucleotide that encodes the polypeptide; (3) an antibody or its fragment that specifically binds to the CCX CKR polypeptide; (4) oligonucleotide or polynucleotide probes and/or primers; (5) CCX CKR inhibitora polynucleotides; and (6) a kit comprising the polypeptides, antibodies and polynucleotides. BIOTECHNOLOGY - Preferred Method: In (M1), the step of contacting involves contacting a cell expressing the polypeptide, fragment or variant. The chemokine and the test compound are labeled, where the label is chosen from a fluorophore, chemiluminescent agent, isotope label and an enzyme or its combination. The CCX CKR polypeptide, fragment or variant is a portion of a cell fraction. The chemokine is ELC, SLC, %TECK%, BLC, CTACK, mMIP-1gamma or vMIPII. The variant has at least 95% or 98% sequence identity to SEQ ID No. 2. ACTIVITY - Antiinflammatory; Antiallergic; %Gastrointestinal%-Gen.; Antipsoriatic; Immunosuppressive; Antiarthritic; Antirheumatic; Neuroprotective; Cytostatic; Ophthalmological; Antimicrobial. No supporting data is given. MECHANISM OF ACTION - Modulates binding of CCX CKR polypeptide to chemokine (claimed). USE - (M1) is useful for identifying a modulator of the binding of CCX CKR polypeptide to a chemokine (claimed). The modulator identified by (M1) is useful for treating CCX CKR-mediated diseases and conditions, such as inflammatory or allergic disease (e.g. systemic anaphylaxis or hypersensitivity responses, inflammatory %bowel% disease, psoriasis and drug allergies), autoimmune diseases (e.g. rheumatoid arthritis and multiple sclerosis), graft rejection, cancers, neoplastic disease, retinopathy, macular degeneration, infectious disease and immunosuppressive disease. The modulator is useful for isolating receptor mutants that are excellent screening tools for more potent compounds, for establishing or determining the binding site of other compounds to the CCX CKR chemokine receptor, and for evaluating putative specific modulator of the CCX CKR chemokine receptor, relative to other chemokine receptors. ADMINISTRATION - The modulator is administered at a dosage of 0.001-100 (preferably 0.05-10) mg/kg body weight, by oral, intramuscular, intraperitoneal, intravenous, intracisternal, intracerebroventricular, subcutaneous, nasal, vaginal, sublingual or topical route, or by inhalation. EXAMPLE - Identification of small molecule modulator (compound I, II and III) of CCX CKR polypeptide, was carried out as follows. Source plates of chemical libraries were obtained from commercial vendors and contained individual compounds (5 or 1 mg/ml) in dimethylsulfoxide (DMSO). Multiple compound plates containing 10 compound in each well were prepared, and these were diluted in 20% DMSO to a concentration of 50 micrograms/ml. An aliquot of each mixture (20 mul) was added to the test plates. Human embryonic kidney 293 (HEK293) cells stably expressing the M1 flag epitope-tagged CCX CKR were cultured in Dulbecco's modified Eagle medium (DMEM)-10% fetal bovine serum (FBS), and harvested when the concentration was 0.5-1x106 cells/ml. The cells were centrifuged and resuspended in assay buffer (containing (in mM) N-(2-hydroxyethyl)piperazine-N'-ethanesulfonic acid (HEPES) (20), (80) sodium chloride, (1) calcium chloride, (5) magnesium chloride and with 0.2% bovine serum albumin, pH 7.4) to a concentration of 5.6x106 cells/ml. Using a multi-probe automated system, the cells (0.09 ml) was added to each well of the assay test plates containing the compounds, followed by 125 I-MIPbeta-3/ELS (0.09 ml) diluted in assay buffer. The final concentration of the compounds was 1-5 micrograms/ml each. The plates were sealed and incubated for approximately 3 hours at 4degreesC on a shaker platform. The assay plates were harvested. Scintillation fluid (50 mul) was added to each of the wells, the plates were sealed and counted in a Top Count scintillation counter. Control wells contained either diluent only or excess EBI-1-ligand chemokine (ELC) (1 micrograms/ml, for non-specific binding) and were used to calculate the percent of total inhibition of ELC binding for each set of compounds. The compounds I and II were found to inhibit binding between ELC and CCX CKR. Compound III was determined to enhance binding.(43 pages)

2/7/93 (Item 2 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0379377 DBR Accession No.: 2005-25083 PATENT
New antibody, or its fragment, that binds a mammalian %TECK% and inhibits binding of the %TECK% to a mammalian GPR-9-6, useful for diagnosing, preventing or treating inflammatory diseases or cancer, or in drug screening purposes - humanized antibody production against human %TECK% for use in disease diagnosis and therapy
AUTHOR: ANDREW D P; ZABEL B A; PONATH P D
PATENT ASSIGNEE: MILLENNIUM PHARM INC 2005
PATENT NUMBER: US 20050181501 PATENT DATE: 20050818 WPI ACCESSION NO.: 2005-590382 (200560)
PRIORITY APPLIC. NO.: US 109349 APPLIC. DATE: 20050419
NATIONAL APPLIC. NO.: US 109349 APPLIC. DATE: 20050419
LANGUAGE: English
ABSTRACT: DERWENT ABSTRACT: NOVELTY - An antibody, or its antigen-binding fragment, that binds a mammalian %TECK% and inhibits binding of the mammalian %TECK% to a mammalian GPR-9-6, is new. DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for: (1) an immunoconjugate comprising an antibody or its antigen-binding portion that is directly or indirectly bonded to an additional therapeutic agent, where the antibody or its antigen-binding portion binds a mammalian %TECK% and inhibits binding of the mammalian %TECK% to a mammalian GPR-9-6; (2) an antigen-binding fusion protein comprising an antigen-binding portion of an antibody that is directly or indirectly bonded to an additional therapeutic agent, where the antigen-binding portion binds a mammalian %TECK% and inhibits binding of the mammalian %TECK% to a mammalian GPR-9-6, and where the antigen-binding portion and the additional therapeutic agent are part of a contiguous polypeptide; (3) an isolated cell that produces the above-mentioned antigen-binding fusion protein, or antibody or its antigen-binding fragment; (4) treating a subject having an inflammatory disease; (5) detecting a mammalian %TECK% or its portion in a biological sample; and (6) a test kit for detecting mammalian %TECK% or its portion in a biological sample, comprising at least one antibody or its antigen-binding fragment cited above; and one

or more ancillary reagents suitable for detecting the presence of a complex between the antibody or its antigen-binding fragment and the mammalian %TECK% or portion. WIDER DISCLOSURE - (1) detecting or identifying an agent that binds to a mammalian GPR-9-6; and (2) treating a subject having cancer. BIOTECHNOLOGY - Preferred Antibody: The mammalian %TECK% is human %TECK%. The human %TECK% is encoded by a sequence having 879 or 876 bp fully defined in the specification (SEQ ID NO: 8 or 10). The mammalian GPR-9-6 is human GPR-9-6, and is encoded by the open reading frame of a sequence having 2577 bp fully defined in the specification (SEQ ID NO: 1). The antibody or antigen-binding fragment inhibits a function mediated by mammalian GPR-9-6 in response to %TECK% binding. The function mediated by mammalian GPR-9-6 is %TECK% -induced chemotaxis. The binding of the antibody or antigen-binding fragment to the mammalian %TECK% is inhibited by mAb 11.3.1 or mAb 16.3.1. The antibody or antigen-binding fragment has the epitopic specificity of mAb 11.3.1 or mAb 16.3.1. The antibody or antigen-binding fragment is selected from a human antibody, an antigen-binding fragment of a human antibody, a humanized antibody, an antigen-binding fragment of a humanized antibody, a chimeric antibody, and an antigen-binding fragment of a chimeric antibody. In addition, the antibody or antigen-binding fragment is the antibody produced by murine hybridoma 11.3.1 or 16.3.1, or its antigen-binding fragment. Preferred Cell: The isolated cell is murine hybridoma 11.3.1 or 16.3.1. Preferred Method: Treating a subject having an inflammatory disease comprises administering to the subject an amount of the antibody or its antigen-binding fragment cited above. Detecting a mammalian %TECK% or its portion in a biological sample comprises contacting a biological sample with the above antibody or antigen-binding fragment under conditions for binding of the antibody or antigen-binding fragment to a mammalian %TECK% ; and detecting binding of the antibody or its antigen-binding fragment, where binding of the antibody or antigen-binding fragment indicates the presence of the %TECK% or its portion. ACTIVITY - Antiinflammatory; %Gastrointestinal% -Gen.; Cytostatic. No biological data given. MECHANISM OF ACTION - Gene Therapy. USE - The composition and methods are useful for diagnosing, preventing or treating inflammatory diseases (e.g. inflammatory %bowel% disease) or cancer. These may also be used in screening for agents that may treat or prevent such diseases. ADMINISTRATION - Dosages may range from about 0.01-100 mg/kg of body weight. Administration can be oral, topical, transdermal, rectal, intravenous, intraarterial, intramuscular, subcutaneous, intrathecal, intradermal, inhalational, and the likes. EXAMPLE - No suitable example given.(62 pages)

2/7/94 (Item 3 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0378422 DBR Accession No.: 2005-24128 PATENT
Inhibiting GPR-9-6 function for research, therapeutic, prophylactic or diagnostic purposes comprises contacting a cell that expresses a mammalian GPR-9-6 with an antibody that binds GPR-9-6 and blocks binding of %TECK% to the GPR-9-6 - production of recombinant G-protein coupled receptor-9-6 useful for the generation of a monoclonal antibody useful for a cancer drug screening application
AUTHOR: ANDREW D P; ZABEL B A; PONATH P D
PATENT ASSIGNEE: MILLENNIUM PHARM INC 2005
PATENT NUMBER: US 6936248 PATENT DATE: 20050830 WPI ACCESSION NO.: 2005-568998 (200558)
PRIORITY APPLIC. NO.: US 522752 APPLIC. DATE: 20000310
NATIONAL APPLIC. NO.: US 522752 APPLIC. DATE: 20000310
LANGUAGE: English
ABSTRACT: DERWENT ABSTRACT: NOVELTY - Inhibiting a function of G protein receptor GPR-9-6 comprises contacting a cell expressing a mammalian GPR9-6 with an antibody, or its antigen-binding fragment, which binds the mammalian GPR-9-6 and inhibits binding of %TECK% (thymus-expressed chemokine) to the mammalian GPR-9-6. WIDER DISCLOSURE - Also disclosed are: (a) a method for detecting or identifying an agent that binds to a mammalian GPR-9-6; (b) methods for detecting or quantifying a mammalian GPR-9-6 or %TECK% , or their portions, in a biological sample; (d) a test kit for identifying or quantifying a mammalian GPR-9-6 or its portion in a biological sample; and (d) methods for treating a subject having cancer or inflammatory disease. BIOTECHNOLOGY - Preferred Method: The antibody or antigen-binding fragment binds the GPR-9-6 of SEQ ID NO:2 (357 amino acids), is inhibited in binding to the GPR-9-6 by a peptide comprising SEQ ID NO:3 (26 amino acids), by mAb 3C3 (ATCC Accession No. HB-12653), or by mAb GPR96-1 (ATCC Accession No. PTA-1470), and/or has the epitopic specificity of mAb 3C3 or mAb GPR96-1. The mammalian GPR-9-6 is a human GPR-9-6. The cell is a recombinant cell line, preferably MOLT-4 or MOLT-13. The cell is a primary (T) cell. The cell is contacted with the antibody or antigen-binding fragment in vitro or in vivo. The GPR-9-6 is encoded by SEQ ID NO:1 (2577 bp). The antibody or antigen-binding fragment is selected from a human antibody, a humanized antibody, a chimeric antibody, and their antigen-binding fragments. The antigen-binding fragment is selected from a Fab fragment, a Fab' fragment, an F(ab')2 fragment, and an Fv fragment. ACTIVITY - Cytostatic; %Gastrointestinal% -Gen.; Antiinflammatory. No biological data given. MECHANISM OF ACTION - GPR-9-6-Inhibitor. USE - The methods and composition are useful for inhibiting the function of GPR-9-6 for research, therapeutic, prophylactic or diagnostic purposes, particularly for treating diseases such as cancer or inflammatory %bowel% disease. They may also be used in drug screening purposes. ADMINISTRATION - Antibody dosages may range from about 0.01-100 mg/kg of body weight. Administration can be oral, topical, transdermal, rectal, intravenous, intraarterial, intramuscular, subcutaneous, intrathecal, intradermal, inhalational, and the like. EXAMPLE - No relevant example given.(62 pages)

2/7/95 (Item 4 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0318419 DBR Accession No.: 2003-19559 PATENT
Treating cancer, involves administering tumor-derived dendritic cell inhibitory factor antagonist in combination with Toll-like receptor agonist, to an individual in need of the treatment - recombinant antagonist and agonist for administration in cancer mouse animal model for cancer therapy
AUTHOR: VICARI A P; CAUX C
PATENT ASSIGNEE: SCHERING CORP 2003
PATENT NUMBER: WO 200345431 PATENT DATE: 20030605 WPI ACCESSION NO.: 2003-493377 (200346)
PRIORITY APPLIC. NO.: US 333434 APPLIC. DATE: 20011127
NATIONAL APPLIC. NO.: WO 2002US38098 APPLIC. DATE: 20021126
LANGUAGE: English
ABSTRACT: DERWENT ABSTRACT: NOVELTY - Treating cancer, involves administering an effective amount of a tumor-derived dendritic cell (DC) inhibitory factor antagonist (I) in combination with an effective amount of Toll-like receptor (TLR) agonist (II), to an individual in need of the treatment. BIOTECHNOLOGY - Preferred Method: (I) is selected from antagonists of interleukin (IL)-6, vascular endothelial growth factor (VEGF), cytotoxic T lymphocyte antigen, Cytotoxic T-lymphocyte associated molecule-4 (CTLA-4), OX-40, transforming growth factor (TGF)-B, prostaglandin, ganglioside, macrophage-colony stimulating factor (M-CSF) and IL-10 or IL-10 receptor. IL-10 antagonist is recombinant, a natural ligand, small molecule, an antibody or its fragment, antisense nucleotide sequence or a soluble IL-10 receptor molecule. (II) is recombinant, a natural ligand, immunostimulatory nucleotide sequence, small molecule, purified bacterial extract or an inactivated bacterial preparation. (II) is TLR-9 or an immunostimulatory nucleotide sequence e.g. CpG motif selected from CpG 2006 (TCGTCGTTTGTCGTTTTGTCGTT), CpG 2216 (GGGGGACGATCGTCGGGGGG), AAC-30 (ACCGATAACGTTGCCGGTGACGGCACCACG) and GAC-30 (ACCGATGACGTCGCCGGTGACGGCACCACG). The immunostimulatory nucleotide molecule is stabilized by structure modification such as phosphorothioate-modification, or is encapsulated in cationic liposomes. The method further involves administering a substance which allows for slow release of (I) and (II) at a delivery site, and at

least one tumor-associated antigen linked to TLR agonist. The tumor-associated antigen is selected from any one of the compounds given in the specification, e.g. Melan-A, tyrosinase, p97 and high molecular weight melanoma antigen. The method further involves administering an activating agent e.g. interferon (IFN)-alpha, TNFalpha, RANK ligand/agonist, CD40 ligand/agonist, or a ligand/agonist of another member of the TNF/CD40 receptor family. The method further involves delivering a chemokine (e.g. CCL21, CCL3, CCL20, CCL16, CCL5, %CCL25% , CXCL12, CCL17, CCL8, CCL2, CCL13, CXCL9, CXCL10 and CXCL11) active on dendritic cells, to the tumor. The chemokine is delivered to the tumor using a targeting construct comprising a chemokine or a biologically active fragment or its variant, and a targeting moiety. The targeting moiety is selected from a peptide of at least 10 amino acids, a protein, a small molecule, a vector or an antibody or its fragment. (I) and (II) are linked to each other, and further linked to a tumor associated antigen. ACTIVITY - Cytostatic. C26-6CK tumor cells (1x105 cells) were implanted subcutaneously at Day 0 in groups of seven 8-week old female BALB/c mice. Ten mug of CpG 1668 was injected peri- (when tumor too small) or intratumorally at Day 7,14 and 21. Anti-IL-10R purified antibody (250 mug) was injected intraperitoneally twice a week starting at Day 7 (stop Day 24). Control antibody was purified GL113 antibody. Tumor development was assessed three times a week by palpation and tumors measured using calipers with tumor volume = I2xLx0.4, where I is the small diameter and L is the large diameter. Mice were sacrificed when tumors exceeded 1500 mm3 or more for human criteria. The results showed that all the mice injected with control antibody or anti-IL-10R antibody alone developed tumors within 7-10 days, that eventually led to the sacrifice of animals at around 4 weeks. Injection of the TLR-9 agonist CpG 1668 had a minor effect since 1/7 mouse did not develop a tumor. In addition, survival was slightly better than this CpG 1668 group and the mean volume of tumors smaller than in the control group after 3 weeks. In contrast, mice treated with the combination of CpG 1668 and anti-IL-10R, although developing palpable tumors, rejected these tumors for 6 out of 7 mice. Subsequently, those mice remained tumor-free for the rest of the experiment. The results indicated that the combination of TLR-9 agonist and IL-10 antagonist has therapeutic value in the C26-6CK model, suggesting that it could be used to treat other tumors, including in man. MECHANISM OF ACTION - Activator of dendritic cells that are rendered hypo-responsive to activation stimuli by the disease. USE - The method is useful for treating cancer e.g. melanoma, breast, pancreas, %colon%, lung, glioma, hepatocellular, endometrium, gastric, %intestinal% , renal, prostate, thyroid, ovarian, testicular, liver, head and neck, colorectal, esophagus, stomach, eye, bladder, glioblastoma and metastatic carcinomas (claimed). ADMINISTRATION - (I) and (II) are administered through intravenous, intratumoral, intradermal, intramuscular, subcutaneous or topical route (claimed). Dosage of (I) is 0.05-25 microg/kg/day, preferably 1-10 microg/kg/day, and dosage of (II) is 0.1-100 microg. (47 pages)

2/7/96 (Item 5 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0317359 DBR Accession No.: 2003-18499 PATENT
A protein useful for prevention and/or treatment of inflammatory %intestinal% diseases - recombinant protein production and antibody useful for disease therapy
PATENT ASSIGNEE: TEIJIN LTD 2002
PATENT NUMBER: JP 2002345487 PATENT DATE: 20021203 WPI ACCESSION NO.: 2003-460883 (200344)
PRIORITY APPLIC. NO.: JP 2000268647 APPLIC. DATE: 20000905
NATIONAL APPLIC. NO.: JP 2001263101 APPLIC. DATE: 20010831
LANGUAGE: Japanese
ABSTRACT: DERWENT ABSTRACT: NOVELTY - A protein consisting of a fully defined 61 amino acid sequence (P1) given in the specification, is new. DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for: (1) a protein consisting of an amino acid sequence in which at least one amino acid is replaced, deleted or added in the amino acid sequence of P1 and competitively inhibiting specifically the combination of chemokine %TECK% to CCR9 receptor; (2) a DNA encoding the above protein; (3) a DNA hybridizing with the above DNA and encoding a protein competitively inhibiting specifically the combination of chemokine %TECK% to CCR9 receptor; (4) a protein prepared by expressing the above DNA; (5) a vector containing the above DNA; (6) a host in which the above vector is recombined; (7) an antibody against the above protein; (8) selecting a substance inhibiting specifically the combination of the above protein to CCR9 receptor by using the above protein as the labelling ligand; (9) a substance selected by the above method; (10) and a drug composition containing the above protein as the active component. USE - The protein is useful as a biochemical reagent or a treating and/or preventing agent for inflammatory %intestinal% diseases such as diabrotic colitis and %Crohn%'s disease. (9 pages)

2/7/97 (Item 6 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0306109 DBR Accession No.: 2003-07894 PATENT
New antibody or its antigen-binding fragment that binds to a mammalian GPR-6 and inhibits the binding of a ligand to the GPR-9-6, useful for treating inflammatory disease e.g., inflammatory %bowel% disease - monoclonal antibody production by hybridoma cell culture useful for inflammatory disease gene therapy
AUTHOR: ANDREW D P; ZABEL B A; PONATH P D
PATENT ASSIGNEE: MILLENNIUM PHARM INC 2002
PATENT NUMBER: US 20020141991 PATENT DATE: 20021003 WPI ACCESSION NO.: 2003-165809 (200316)
PRIORITY APPLIC. NO.: US 759 APPLIC. DATE: 20011023
NATIONAL APPLIC. NO.: US 759 APPLIC. DATE: 20011023
LANGUAGE: English
ABSTRACT: DERWENT ABSTRACT: NOVELTY - Antibody or its antigen-binding fragment which binds to a mammalian GPR-6 and inhibits the binding of a ligand to the GPR-9-6, is new. DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for: (1) an isolated cell that produces the antibody or its antigen-binding fragment; (2) detecting a mammalian GPR-9-6; (3) detecting and identifying an agent that binds to, or that is an inhibitor of, a mammalian GPR-9-6 or its ligand binding variant; (4) treating a subject having an inflammatory disease or treating an inflammatory %bowel% disease; (5) inhibiting GPR-9-6-mediated homing of leukocytes to mucosal tissue in a subject; (6) modulating a GPR-9-6 function; (7) a test kit for detecting the presence of a mammalian GPR-9-6 or %TECK% or its portion in a biological sample; and (8) detecting a mammalian %TECK% or its portion in a biological sample.BIOTECHNOLOGY - Preferred Antibody: The antibody or its antigen-binding fragment binds to the mammalian %TECK%, GPR-9-6 or a similar epitope as mAb 3C3 or mAb GPR-9-6. It is produced by murine hybridoma 3C3, 11.3.1 or 16.3.1. It consists of: (1) mAb 3C3 or mAb GPR-9-6; (2) an antibody that can compete with mAb 3C3 or mAb GPR-9-6-1 for binding to a mammalian a GPR-9-6; and/or (3) antigen-binding fragments of (1) or (2) that bind a mammalian GPR-9-6 or its portion. The mammalian %TECK% is human %TECK%. The mammalian GPR-9-6 is human GPR-9-6. The ligand is %TECK% . The binding of antibody or its antigen-binding fragment to GPR-6 can be inhibited by a peptide that consists of the 26-amino acid sequence or by mAb 3C3 or mAb GPR96-1. Preferred Cell: The isolated cell consists of an immortalized B cell, a hybridoma or a recombinant cell comprising one or more exogenous nucleic acid molecules that encode the antibody or its antigen-binding fragment. Preferred Method: Detecting a mammalian GPR-9-6 comprises: (1) contacting a biological sample with the antibody or its antigen-binding fragment for binding of the antibody or its antigen-binding fragment to a mammalian GRP-9-6 or its portion; and (2) detecting binding of the antibody or its antigen-binding fragment, where the binding of the antibody or its antigen-binding fragment indicates the presence of the receptor or its portion. The biological sample is of human origin. Detecting and identifying an agent that binds to a mammalian GPR-9-6 or its ligand binding variant comprises: (1) combining a reference agent, a test agent and a composition

comprising a functional mammalian GPR-9-6 or its ligand binding variant; (2) detecting and measuring the formation of a complex between the reference agent and the GPR-9-6 or its ligand binding variant, where a decrease in the formation of the complex relative to a control indicates that the test agent binds to the GPR-9-6 or its ligand binding variant. The reference agent is labeled with a label comprising radioisotope, epitope, affinity label, enzyme, fluorescent group or chemiluminescent group. The composition is a cell that expresses a mammalian GPR-9-6, or a membrane preparation of a cell that expresses a mammalian GPR-9-6 or its ligand binding variant. The cell is a recombinant cell or a cell line. It comprises MOLT-4 or MOLT-13. Detecting and identifying an agent that is an inhibitor of a mammalian GPR-9-6 receptor comprises: (1) combining an agent to be tested, a ligand or promoter of the GPR-9-6 and a cell expressing the GPR-9-6 for detecting a ligand- or promoter-induced response; and (2) determining the ability of the test compound to inhibit the response, where inhibition of a ligand- or promoter-induced response by the agent indicates that the agent is an inhibitor. The response is chemotaxis or Ca2- flux. Inhibiting GPR-9-6-mediated homing of leukocytes to mucosal tissue in a subject or treating a subject having an inflammatory disease or treating an inflammatory %bowel% disease comprises administering an antagonist of a mammalian GPR-9-6 function to the subject. The inflammatory disease is %Crohn%'s disease or colitis. Modulating a GPR-9-6 function comprises contacting a cell that expresses GPR-9-6 with an agent that binds to it. The agent can inhibit a function of GPR-9-6. It is an antibody or antigen-binding fragment. The function consists of ligand binding, ligand-induced chemotaxis or ligand-induced Ca2- flux. The antagonist inhibits the binding of the ligand to the mammalian GPR-9-6. Detecting a mammalian %TECK% or its portion in a biological sample comprises: (1) contacting a biological sample with the antibody or its antigen-binding fragment for binding of the antibody or its antigen-binding fragment to a mammalian a %TECK% or its portion; and (2) detecting binding of the antibody or its antigen-binding fragment, where the binding of the antibody or its antigen-binding fragment indicates the presence of the receptor or its portion. Preferred Test Kit: The test kit for detecting the presence of GPR-9-6 comprises: (1) at least one antibody or its antigen-binding fragment that binds to a mammalian GPR-9-6 or its portion; or (2) one or more ancillary reagents for detecting the presence of a complex between the antibody or its antigen-binding fragment and the mammalian GPR-9-6 or its portion. The test kit for detecting the presence of mammalian %TECK% comprises: (1) at least one antibody or its antigen-binding fragment that binds to a mammalian %TECK% or its portion; or (2) one or more ancillary reagents for detecting the presence of a complex between the antibody or its antigen-binding fragment and the mammalian %TECK% or its portion. ACTIVITY - Antiinflammatory. No biological data given. MECHANISM OF ACTION - GPR-6-Inhibitor; Gene therapy. USE - The antibody is useful for treating a subject having an inflammatory disease or treating an inflammatory %bowel% disease (claimed). ADMINISTRATION - Dosage comprises 0.01-100 mg/kg body weight. The composition may be administered via oral, topical, rectal or parenteral route. EXAMPLE - No relevant examples given. (64 pages)

2/7/98 (Item 7 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0294123 DBR Accession No.: 2002-15970 PATENT
Using chemokine MCP-4 or 6Ckine to attract dendritic cells to the site of an antigen is useful to treat disease states, particularly autoimmune disease, tissue rejection, allergy and cancer - mammal immune response enhancement for disease therapy
AUTHOR: VICARI A P; CAUX C; LAFACE D
PATENT ASSIGNEE: VICARI A P; CAUX C; LAFACE D 2002
PATENT NUMBER: US 20020034494 PATENT DATE: 20020321 WPI ACCESSION NO.: 2002-351086 (200238)
PRIORITY APPLIC. NO.: US 768917 APPLIC. DATE: 20010124
NATIONAL APPLIC. NO.: US 768917 APPLIC. DATE: 20010124
LANGUAGE: English
ABSTRACT: DERWENT ABSTRACT: NOVELTY - Enhancing an immune response in a mammal, comprising administering chemokine MCP-4 or 6Ckine or their biologically active fragment, is new. DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for: (1) fusion proteins comprising the chemokine MCP-4 or 6Ckine and an antigen; (2) a plasmid comprising the fusion protein, preferably further comprising a promoter sequence suited for dendritic cells; (3) a viral vector comprising one of the fusion proteins; (4) use of a chemokine capable of directing the migration of dendritic cells to manufacture a medicament for a disease state. BIOTECHNOLOGY - Preferred Method: When enhancing an immune response the chemokine is preferably recombinant and human, and is administered alongside a substance which allows for the slow release of the chemokine at the delivery site. Preferably the method further comprises administering an antigen with the chemokine, or a fusion protein of the chemokine and antigen. The antigen is preferably tumor-associated antigen or a bacterial, viral or fungal-associated antigen. The tumor-associated antigen is preferably Melan-A, tyrosinase, p97, beta-HCG, GalNAc., melanoma associated neoplastic protein-1 (MAGE-1), MAGE-2, MAGE-3, MAGE-4, MAGE-12, MART-1, MUC1, MUC2, MUC3, MUC4, MUC18, CEA, DDC, melanoma antigen gp75, Hker8, high molecular weight melanoma antigen, K19, Tyr1, Tyr2, a member of the pMel 17 gene family, c-Met, PSA, PSM, alpha-fetoprotein, thyroperoxidase, gp 100, p53 or telomerase, or with 6Ckine, C26 %colon% carcinoma. Preferably the method further comprises administering a combination of GM-CSF and IL4 and/or an activating agent. When manufacturing a medicament the chemokine is preferably MCP-1, MCP-2, MCP-3, MCP-4, macrophage inflammation protein-1alpha (MIP-1alpha), MIP-1-beta, MIP-3alpha, RANTES, SDF-1, %Teck% , DCtactin-beta, 6CKine/SLC, LEC, MDC or MIP-5. The chemokine is preferably capable of directing migration of dendritic cells to a site of antigen delivery or to lymphoid organs. Preferably the dendritic cells are immature, and the chemokine is MCP-1,2,3 or 4, MDC, RANTES, , MIP-1alpha, MIP-1-beta, MIP-3alpha or MIP-5. Where the cells are directed to lymphoid organs the cytokine is preferably MIP-3alpha. The disease for which medicament is manufactured is preferably an autoimmune disease, tissue rejection or an allergy, or a cancer, particularly melanoma, breast, pancreatic, %colon%, lung, glioma, hepatocellular, endometrial, gastric, %intestinal% , renal, prostate, thyroid, ovarian, testicular, liver, head and neck, colorectal, esophagus, stomach, eye or bladder cancer, glioblastoma or metastatic carcinomas. Preferably manufacture further comprises using at least one disease-associated antigen, preferably a tumor-associated or bacterial, viral or fungal antigen. The tumor-associated antigen is preferably one of those described above, more preferably where the cancer is prostate cancer and the antigen is PSA and/or PSM, or melanoma, and the antigen is Melan-A, gp100 or tyrosinase. An activating agent, more preferably TNF-alpha, RP-105, an anti-CD-40 antibody or nucleic acids containing unmethylated CpG motifs or ligands or toll-like receptors is preferably also used, plus a combination of granulocyte-macrophage colony stimulating factor (GM-CSF) and interleukin-4(IL-4). ACTIVITY - Immunosppressive; Antiallergic; Cytostatic. cDNA encoding mouse 6Ckine/SLC was cloned into the pcDNA3 vector which contains a CMV promoter. C26 %colon% carcinoma cells were transfected with this construction using Fugene reagent, and single C26 clones expressing m6Ckine/SLC mRNA were obtained after neomycin selection at 800 microg/ml. C26 or C26-6CK tumor cells in 100 microl Dulbecco's modified Eagle's medium (DMEM) were injected subcutaneously in the right flank of 6-10 week old female BALB/c mice and tumor growth was monitored by palpitation 3 times a week. Tumors were surgically removed when the size reached approximately 1 cm. The mass was minced into small fragments and incubated in collagenase A for 30 mins at 37 degrees C under agitation, washed in DMEM and stained in phosphate buffered saline (PBS) + 5% fetal calf serum (FCS). Prior to incubation with FITC-, biotin or PE-labeled specific antibodies, Fc receptors were blocked using Fc-Block (RTM) CD16/CD32 antibody (Pharmingen). The various antibodies used were CD8beta, CD11c, anti-MHC class II-Ad/I-Ed, and CD3 (Pharmingen). C26 wild-type tumors and or C26-6CK tumors expressing m6Ckine were analyzed for CD8 T cells and CD11c+MHC class II+ dendritic cell infiltration by flow cytometry analysis of whole tumor suspension

(n=7). Data show a significant recruitment of both leukocyte subsets in C26-CK tumors compared to C26 tumors (see figure). These results suggest that m6Ckine gene transfer into tumors promotes the recruitment of CD8 T and dendritic cells. MECHANISM OF ACTION - Dendritic cell migration attractant. USE - The invention is used to treat disease states, including an autoimmune disease, tissue rejection or an allergy, or a cancer, particularly melanoma, breast, pancreatic, %colon%, lung, glioma, hepatocellular, endometrial, gastric, %intestinal% , renal, prostate, thyroid, ovarian, testicular, liver, head and neck, colorectal, esophagus, stomach, eye or bladder cancer, glioblastoma or metastatic carcinomas (claimed) ADMINISTRATION - When enhancing an immune response, administration is preferably by a method which allows slow release at the delivery site and may be intradermal, intramuscular, subcutaneous, topical or in the form of a vector. No dosage is provided in the source material. EXAMPLE - No suitable example is provided in the source material.(15 pages)

2/7/99 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

144487122 CA: 144(26)487122d JOURNAL
CD8+ recent thymic emigrants home to and efficiently repopulate the small intestine epithelium. (Erratum to document cited in CA144:449323)
AUTHOR(S): Staton, Tracy L.; Habtezion, Aida; Winslow, Monte M.; Sato, Tohru; Love, Paul E.; Butcher, Eugene C.
LOCATION: Program in Immunology, Department of Pathology, Stanford University School of Medicine, Stanford, CA, 94305, USA
JOURNAL: Nat. Immunol. (Nature Immunology) DATE: 2006 VOLUME: 7 NUMBER: 6 PAGES: 672 CODEN: NIAMCZ ISSN: 1529-2908 LANGUAGE: English
PUBLISHER: Nature Publishing Group
SECTION:
CA215010 Immunochemistry
IDENTIFIERS: erratum CD8 lymphocyte homing intestine epithelium
DESCRIPTORS:
Chemokines...
CCL25 (C-C motif ligand 25); CD8+ recent thymic emigrants home to and efficiently repopulate the small intestine epithelium (Erratum)
Chemokine receptors...
CCR9; CD8+ recent thymic emigrants home to and efficiently repopulate the small intestine epithelium (Erratum)
Thymus gland... CD8-positive T cell... Lymphotoxin... Cell differentiation ...
CD8+ recent thymic emigrants home to and efficiently repopulate the small intestine epithelium (Erratum)
Lymphocyte...
homing; CD8+ recent thymic emigrants home to and efficiently repopulate the small intestine epithelium (Erratum)
Integrins...
LPAM-1 (lymphocyte Peyer's patch adhesion mol. 1); CD8+ recent thymic emigrants home to and efficiently repopulate the small intestine epithelium (Erratum)
Cell migration...
lymphocyte homing; CD8+ recent thymic emigrants home to and efficiently repopulate the small intestine epithelium (Erratum)
T cell(lymphocyte)...
proliferation; CD8+ recent thymic emigrants home to and efficiently repopulate the small intestine epithelium (Erratum)
Intestine...
small, epithelium; CD8+ recent thymic emigrants home to and efficiently repopulate the small intestine epithelium (Erratum)
Epithelium...
small intestinal; CD8+ recent thymic emigrants home to and efficiently repopulate the small intestine epithelium (Erratum)
Cell proliferation...
T cell; CD8+ recent thymic emigrants home to and efficiently repopulate the small intestine epithelium (Erratum)

2/7/100 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

144225880 CA: 144(13)225880d JOURNAL
Modulation of immunity-related gene expression in small intestines of mice by oral administration of lactoferrin
AUTHOR(S): Wakabayashi, Hiroyuki; Takakura, Natsuko; Yamauchi, Koji; Tamura, Yoshitaka
LOCATION: Nutritional Science Laboratory, Morinaga Milk Industry Co., Ltd., Zama, Kanagawa, Japan, 228-8583
JOURNAL: Clin. Vaccine Immunol. (Clinical and Vaccine Immunology) DATE: 2006 VOLUME: 13 NUMBER: 2 PAGES: 239-245 CODEN: CVILA6 ISSN: 1556-6811
LANGUAGE: English PUBLISHER: American Society for Microbiology
SECTION:
CA201007 Pharmacology
CA215XXX Immunochemistry
IDENTIFIERS: lactoferrin immunomodulation immunity related gene expression intestine
DESCRIPTORS:
Integrins...
.alpha.4; modulation of immunity-related gene expression in small intestines of mice by lactoferrin
Interferons...
.beta.; modulation of immunity-related gene expression in small intestines of mice by lactoferrin
Chemokines...
CCL25 (C-C motif ligand 25); modulation of immunity-related gene expression in small intestines of mice by lactoferrin
Chemokine receptors...
CCR9; modulation of immunity-related gene expression in small intestines of mice by lactoferrin
Interferons...
.gamma.; modulation of immunity-related gene expression in small intestines of mice by lactoferrin
Proteins...
intelectin; modulation of immunity-related gene expression in small intestines of mice by lactoferrin
Lactoferrins... Gene expression... Immunomodulators... Interleukin 10... Interleukin 18... Interleukin 15...
modulation of immunity-related gene expression in small intestines of mice by lactoferrin
Proteins...
NOD2; modulation of immunity-related gene expression in small intestines of mice by lactoferrin
Interleukin 23...
p19; modulation of immunity-related gene expression in small intestines of mice by lactoferrin
Interleukin 12...
p40; modulation of immunity-related gene expression in small intestines of mice by lactoferrin
Receptors...
TLR-2 (Toll-like receptor-2); modulation of immunity-related gene expression in small intestines of mice by lactoferrin
Receptors...
TLR-3 (Toll-like receptor-3); modulation of immunity-related gene expression in small intestines of mice by lactoferrin
Receptors...
TLR-4 (Toll-like receptor-4); modulation of immunity-related gene expression in small intestines of mice by lactoferrin
5-HT receptors...
type 5-HT1P; modulation of immunity-related gene expression in small intestines of mice by lactoferrin
CAS REGISTRY NUMBERS:
869218-82-6 modulation of immunity-related gene expression in small intestines of mice by lactoferrin
9001-63-2 P; modulation of immunity-related gene expression in small intestines of mice by lactoferrin

2/7/101 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

144086175 CA: 144(6)86175n JOURNAL
CCL25 and CCL28 play essential roles in intestinal homing of IgA antibody-secreting cells
AUTHOR(S): Hieshima, Kunio
LOCATION: Department of Microbiology, Kinki University School of Medicine, Osaka-Sayama, Japan, 589-8511
JOURNAL: Rinsho Men'eki (Rinsho Men'eki) DATE: 2005 VOLUME: 43 NUMBER: 5 PAGES: 584-588 CODEN: RNMKAU ISSN: 0386-9695 LANGUAGE: Japanese PUBLISHER: Kagaku Hyoronsha
SECTION:
CA215000 Immunochemistry
IDENTIFIERS: review chemokine IgA plasma cell mucosal immunity homing
DESCRIPTORS:
Chemokines...
CCL25 (C-C motif ligand 25); chemokines CCL25 and CCL28 in intestinal homing of IgA antibody-secreting cells
Chemokines...
CCL28 (C-C motif ligand 28); chemokines CCL25 and CCL28 in intestinal homing of IgA antibody-secreting cells
Intestine... B cell(lymphocyte)...
chemokines CCL25 and CCL28 in intestinal homing of IgA antibody-secreting cells
Lymphocyte...
homing, plasma cell; chemokines CCL25 and CCL28 in intestinal homing of IgA antibody-secreting cells
Antibodies and Immunoglobulins...
IgA; chemokines CCL25 and CCL28 in intestinal homing of IgA antibody-secreting cells
Immunity...
mucosal; chemokines CCL25 and CCL28 in intestinal homing of IgA antibody-secreting cells
Lymphocyte...
plasma cell; chemokines CCL25 and CCL28 in intestinal homing of IgA antibody-secreting cells

2/7/102 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

144021282 CA: 144(2)21282a JOURNAL
Homing of IgA antibody-secreting cells to the intestinal lamina propria
AUTHOR(S): Hieshima, Kunio; Yoshie, Osamu
LOCATION: School of Medicine, Dep. of Bacteriology, Kinki University, Japan,
JOURNAL: Igaku no Ayumi (Igaku no Ayumi) DATE: 2005 VOLUME: 213 NUMBER: 11 PAGES: 989-993 CODEN: IGAYAY ISSN: 0039-2359 LANGUAGE: Japanese PUBLISHER: Ishiyaku Shuppan
SECTION:
CA215000 Immunochemistry
IDENTIFIERS: review IgA intestine lamina propria chemokine
DESCRIPTORS:
Chemokines...
CCL25 (C-C motif ligand 25); homing of IgA antibody-secreting cells to the intestinal lamina propria
Chemokines...
CCL28 (C-C motif ligand 28); homing of IgA antibody-secreting cells to the intestinal lamina propria
Human...
homing of IgA antibody-secreting cells to the intestinal lamina propria
Antibodies and Immunoglobulins...
IgA; homing of IgA antibody-secreting cells to the intestinal lamina propria
Intestine...
lamina propria; homing of IgA antibody-secreting cells to the intestinal lamina propria
Chemokines...
SDF-1 (stromal-derived factor-1); homing of IgA antibody-secreting cells to the intestinal lamina propria

2/7/103 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

144017169 CA: 144(2)17169b PATENT
Inducers and inhibitors for gut-homing of T-cells, intestinal immunostimulants, manufacture of T-cells with enhanced homing ability, homing-preventing functional foods, and drug screening method
INVENTOR(AUTHOR): Iwata, Makoto; Song, Shih Rong
LOCATION: Japan,
ASSIGNEE: Mitsubishi Chemical Corp.
PATENT: Japan Kokai Tokkyo Koho ; JP 2005336062 A2 DATE: 20051208
APPLICATION: JP 2004153548 (20040524)
PAGES: 18 pp. CODEN: JKXXAF LANGUAGE: Japanese
PATENT CLASSIFICATIONS:
CLASS: A61K-045/00A; A23L-001/303B; A61K-031/203B; A61K-035/14B; A61K-035/26B; A61P-037/04B; A61P-043/00B; C12N-005/06B; C12Q-001/02B; C12N-015/09B
SECTION:
CA201007 Pharmacology
CA215XXX Immunochemistry
CA217XXX Food and Feed Chemistry
IDENTIFIERS: T cell homing intestine immunostimulant retinoic acid, agonist retinoic acid receptor immunostimulant screening, antagonist retinoic acid T cell intestine homing inhibitor, food T cell intestine homing prevention
DESCRIPTORS:
Chemokines...
CCL25 (C-C motif ligand 25), chemotaxis to; retinoic acid receptor agonists/antagonists or cultured T-cells for control of gut-homing of T-cells, immunostimulants, functional foods, and drug screening
Gene,animal...
CCR9, expression; retinoic acid receptor agonists/antagonists or cultured T-cells for control of gut-homing of T-cells, immunostimulants, functional foods, and drug screening method
Chemokine receptors...
CCR9, gene expression; retinoic acid receptor agonists/antagonists or cultured T-cells for control of gut-homing of T-cells, immunostimulants, functional foods, and drug screening method
Cell migration...
homing; retinoic acid receptor agonists/antagonists or cultured T-cells for control of gut-homing of T-cells, immunostimulants, functional foods, and drug screening method
Health food...
low-vitamin A; retinoic acid receptor agonists/antagonists or cultured T-cells for control of gut-homing of T-cells, immunostimulants, functional foods, and drug screening method
Integrins...
LPAM-1 (lymphocyte Peyer's patch adhesion mol. 1), expression; retinoic acid receptor agonists/antagonists or cultured T-cells for control of gut-homing of T-cells, immunostimulants, functional foods,
Immunity...
mucosal; retinoic acid receptor agonists/antagonists or cultured T-cells for control of gut-homing of T-cells, immunostimulants, functional foods, and drug screening method
Immunostimulants... T cell(lymphocyte)... Drug screening... Retinoic acid receptors... CD4-positive T cell... Intestine... Animal tissue culture...
Chemotaxis...
retinoic acid receptor agonists/antagonists or cultured T-cells for control of gut-homing of T-cells, immunostimulants, functional foods, and drug screening method
CAS REGISTRY NUMBERS:
302-79-4 11103-57-4 68-26-8 5300-03-8 94497-51-5 155877-83-1 retinoic acid receptor agonists/antagonists or cultured T-cells for control of gut-homing of T-cells, immunostimulants, functional foods, and drug

screening method
870589-20-1 870589-21-2 870589-22-3 870589-23-4 870589-24-5
870589-25-6 870589-26-7 870589-28-9 870589-29-0 870589-30-3
unclaimed nucleotide sequence; inducers and inhibitors for gut-homing of T-cells, intestinal immunostimulants, manuf. of T-cells with enhanced homing ability, homing-preventing functional foods, and drug screening method

2/7/104 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

143324535 CA: 143(18)324535f DISSERTATION
Expression and regulation of CCL25 and its role in T cell localization and function within the small intestine
AUTHOR(S): Ericsson, Anna Helena
LOCATION: Lunds Universitet, Lund, Swed.
DATE: 2005 PAGES: 161 pp. CODEN: DABBBA LANGUAGE: English CITATION: Diss. Abstr. Int., C 2005, 66(2), 369 AVAIL: From degree-granting institution
SECTION:
CA215005 Immunochemistry
IDENTIFIERS: CCL25 T cell small intestine
DESCRIPTORS:
Chemokines...
CCL25 (C-C motif ligand 25); Expression and regulation of CCL25 and its role in T cell localization and function within the small intestine
T cell(lymphocyte)...
Expression and regulation of CCL25 and its role in T cell localization and function within the small intestine
Intestine...
small; Expression and regulation of CCL25 and its role in T cell localization and function within the small intestine

2/7/105 (Item 7 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

143246765 CA: 143(14)246765q PATENT
Antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
INVENTOR(AUTHOR): Andrew, David P.; Zabel, Brian A.; Ponath, Paul D.
LOCATION: USA
ASSIGNEE: Millennium Pharmaceuticals, Inc.
PATENT: United States ; US 6936248 B1 DATE: 20050830
APPLICATION: US 2000522752 (20000310) *US 266464 (19990311)
PAGES: 62 pp., Cont.-in-part of U.S. Ser. No. 266,464. CODEN: USXXAM
LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: 424143100; A61K-039/40A
SECTION:
CA215003 Immunochemistry
CA201XXX Pharmacology
CA203XXX Biochemical Genetics
CA209XXX Biochemical Methods
CA263XXX Pharmaceuticals
IDENTIFIERS: inflammation cancer diagnosis therapy GPR96 receptor ligand antibody fragment, antibody fragment chemokine receptor GPR96 CCR9 TECK drug screening
DESCRIPTORS:
Antibodies and Immunoglobulins... Drug screening... Immunotherapy... Antitumor agents... Animal cell... T cell(lymphocyte)... Mammalia... Human ... Molecular cloning... DNA sequences... Protein sequences... Inflammation ... Neoplasm...
antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
Chemokines...
C-C, TECK or thymus-expressed chemokine; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
Diagnosis...
cancer; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
Chemokine receptors...
CCR9; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
Antibodies and Immunoglobulins...
chimeric; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
Inflammation...
Crohn's disease; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
Intestine,disease...
Crohn's; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
Antibodies and Immunoglobulins...
fragments; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
Ligands...
GPR-9-6; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
Antibodies and Immunoglobulins...
humanized; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
Diagnosis...
immunodiagnosis; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
Intestine,disease...
inflammatory; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
Animal cell line...
MOLT 13; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
Animal cell line...
Molt 4; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
Antibodies and Immunoglobulins...
monoclonal; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
Leukemia...
T-cell, acute; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
CAS REGISTRY NUMBERS:
863358-38-7P 863358-39-8P 863358-40-1P amino acid sequence; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
294211-61-3 389335-64-2 392015-76-8 antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
863358-35-4P 863358-36-5P 863358-37-6P nucleotide sequence; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer
863362-46-3 863362-47-4 863362-48-5 863362-49-6 863362-50-9 863362-51-0 863362-52-1 863362-53-2 unclaimed nucleotide sequence; antibodies and fragments for inhibiting GPR-9-6 function and for drug screening, diagnosis and treatment of inflammation and cancer

2/7/106 (Item 8 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

143246218 CA: 143(14)246218p JOURNAL

Chemokines in immune responses
AUTHOR(S): Yoshie, Osamu
LOCATION: Department of Microbiology, Kinki University School of Medicine , Japan,
JOURNAL: Saishin Igaku (Saishin Igaku) DATE: 2005 VOLUME: 60 NUMBER: Suppl. PAGES: 626-638 CODEN: SAIGAK ISSN: 0370-8241 LANGUAGE: Japanese
PUBLISHER: Saishin Igakusha
SECTION:
CA215000 Immunochemistry
IDENTIFIERS: review chemokine receptor intestine
DESCRIPTORS:
Chemokines...
CCL25 (C-C motif ligand 25); chemokines and receptors in immune responses of intestine
Chemokines...
CCL28 (C-C motif ligand 28); chemokines and receptors in immune responses of intestine
Chemokine receptors...
CCRL1 (chemokine (C-C motif) receptor-like 1); chemokines and receptors in immune responses of intestine
Chemokine receptors...
CCR6; chemokines and receptors in immune responses of intestine
Chemokine receptors...
CCR9; chemokines and receptors in immune responses of intestine
Macrophage inflammatory protein 3.alpha.... Intestine... Lymphocyte...
chemokines and receptors in immune responses of intestine

2/7/107 (Item 9 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

141048626 CA: 141(4)48626r PATENT
Genes showing altered patterns of expression in response to concentration gradients of cytokines and chemokines
INVENTOR(AUTHOR): Poznansky, Mark; Rutishauser, Rachel
LOCATION: USA
ASSIGNEE: The General Hospital Corp.
PATENT: PCT International ; WO 200453165 A1 DATE: 20040624
APPLICATION: WO 2003US38958 (20031208) *US PV438848 (20030109) *US PV445049 (20030205)
PAGES: 144 pp. CODEN: PIXXD2 LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: C12Q-001/68
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; EG; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NI; NO; NZ; OM; PG; PH; PL; PT; RO; RU; SC; SD; SE; SG; SK; SL; SY; TJ; TM; TN; TR; TT; TZ; UA; UG; US; UZ; VC; VN; YU; ZA; ZM; ZW DESIGNATED REGIONAL: BW; GH; GM; KE; LS; MW; MZ; SD; SL ; SZ; TZ; UG; ZM; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM; AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES; FI; FR; GB; GR; HU; IE; IT; LU; MC; NL; PT; RO; SE; SI; SK; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG
SECTION:
CA203004 Biochemical Genetics
CA215XXX Immunochemistry
IDENTIFIERS: neutrophil chemotaxis fugetaxis chemokine cytokine gene expression
DESCRIPTORS:
Proteins...
actin-capping, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
T cell(lymphocyte)...
activation, genes expressed in, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Macrophage inflammatory protein 2... Interferons...
.alpha., neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
TCR(T cell receptors)...
.alpha. subunit, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Proteins...
AMY-1 (assoc. of Myc-1), gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Proteins...
anaphase promoting complex subunit 10, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Proteins...
angio-assocd. migratory cell protein, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Autoimmune disease... Anemia(disease)...
autoimmune hemolytic anemia, regulating chemotaxis in treatment of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Inflammation... Autoimmune disease... Thyroid gland,disease...
autoimmune thyroiditis, regulating chemotaxis in treatment of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Annexins...
A3, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Glycoproteins...
A33, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Infection...
bacterial, treatment of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Proteins...
BAI-1-assocd., gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokines...
BCA-1, neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Spectrins...
.beta.-, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Transforming growth factors...
.beta.-, neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Transforming growth factor receptors...
.beta.-transforming growth factor, III, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Macrophage inflammatory protein 2... Interferons...
.beta., neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokines...
bolekines, neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Antigens...
bone marrow stromal cell antigen 2, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Skin,disease...
bullous pemphigoid, regulating chemotaxis in treatment of; genes

showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokines...
C-C, HCC-4, neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokines...
C-X-C, GCP-2 (granulocyte chemotactic protein 2), neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokines...
C-X-C, neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokines...
C-10, neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Calcium-binding proteins...
calgranulin A, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Calcium-binding proteins...
calgranulin C, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Cell adhesion molecules...
carcinoembryonic antigen-related 3, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Glycoproteins...
cartilage GP39, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokines...
CCL1 (C-C motif ligand 1), neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokines...
CCL14 (C-C motif ligand 14), neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokines...
CCL15 (C-C motif ligand 15), neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokines...
CCL17 (C-C motif ligand 17), neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokines...
CCL18 (C-C motif ligand 18), neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokines...
CCL25 (C-C motif ligand 25), neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Gene,animal...
CDC27, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Gene,animal...
CDC42, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
CD antigens...
CD24, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Glycoproteins...
CD40-L (antigen CD40 ligand), neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Human...
cells of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Neutrophil...
chemotaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Neutrophil...
chemotaxis; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Transcription factors...
CIITA (class II transactivator), gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Proteins...
complexes, Arp23, 20kDa subunit, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Inflammation...
Crohn's disease, regulating chemotaxis in treatment of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Intestine,disease...
Crohn's, regulating chemotaxis in treatment of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokines...
CTAK, neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokine receptors...
CXCR5, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokine receptors...
CX3CR1, neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Proteins...
cytoskeleton-assocd., gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Proteins...
DAB2 (disabled 2), gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Fertility...
disorder, autoimmune, regulating chemotaxis in treatment of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Immunity...
disorder, regulating chemotaxis in treatment of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokines...
ENA-78, neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Antigens...
epithelial V-like 1, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Autoimmune disease...
exptl. autoimmune encephalomyelitis, regulating chemotaxis in treatment of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Encephalomyelitis...
exptl. autoimmune, regulating chemotaxis in treatment of; genes showing altered patterns of expression in response to concn. gradients of

cytokines and chemokines
Proteins...
extracellular matrix-assocd., gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Proteins...
FGF receptor activating protein 1, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Proteins...
fibulin 1, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Proteins...
ficolins, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Hemopoietins...
FLT3 ligand, neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Proteins...
focal adhesion kinase-assocd. GTPase-regulating, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
Chemokines...
fractalkines, neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
CAS REGISTRY NUMBERS:
9004-10-8 biological studies, resistance, regulating chemotaxis in treatment of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
158129-99-8 444993-55-9 153190-47-7 196717-98-3 144114-16-9 203810-05-3 170347-45-2 9032-64-8 115926-52-8 169592-62-5 146702-84-3 37289-25-1 9012-42-4 90698-26-3 141436-78-4 125978-95-2 104645-76-3 9001-12-1 271597-12-7 150605-50-8 90119-11-2 gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
37270-94-3 65154-06-5 80295-54-1 71160-24-2 89663-86-5 83869-56-1 143011-72-7 81627-83-0 127464-60-2 21581-37-3 116243-73-3 neutrophil chemotaxis in response to; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines
302355-88-0 Receptor protein tyrosine phosphatase, gene for, expression in fugetaxis of; genes showing altered patterns of expression in response to concn. gradients of cytokines and chemokines

2/7/108 (Item 10 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

139322183 CA: 139(21)322183h JOURNAL
Selective generation of gut tropic T cells in gut-associated lymphoid tissue (GALT): Requirement for GALT dendritic cells and adjuvant
AUTHOR(S): Johansson-Lindbom, Bengt; Svensson, Marcus; Wurbel, Marc-Andre ; Malissen, Bernard; Marquez, Gabriel; Agace, William
LOCATION: Immunology Section, Department of Cell and Molecular Biology, Lund University, S-22184, Lund, Swed.
JOURNAL: J. Exp. Med. (Journal of Experimental Medicine) DATE: 2003
VOLUME: 198 NUMBER: 6 PAGES: 963-969 CODEN: JEMEAV ISSN: 0022-1007
LANGUAGE: English PUBLISHER: Rockefeller University Press
SECTION:
CA215010 Immunochemistry
IDENTIFIERS: T cell homing GALT CCR9 receptor
DESCRIPTORS:
T cell(lymphocyte)...
activation; requirement for GALT-derived dendritic cells in T-cell activation, CCR9 receptor induction, and homing to small intestine
Chemokines...
CCL25; requirement for GALT-derived dendritic cells in T-cell activation, CCR9 receptor induction, and homing to small intestine
Receptors...
CCR9; requirement for GALT-derived dendritic cells in T-cell activation, CCR9 receptor induction, and homing to small intestine
Lymph node...
dendritic cell; requirement for GALT-derived dendritic cells in T-cell activation, CCR9 receptor induction, and homing to small intestine
Lipopolysaccharides...
Escherichia coli; homing of T-cells to intestine in relation to GALT-derived dendritic cell maturation in response to
Lymphatic system...
gut-assocd.; requirement for GALT-derived dendritic cells in T-cell activation, CCR9 receptor induction, and homing to small intestine
Lymphocyte...
homing; requirement for GALT-derived dendritic cells in T-cell activation, CCR9 receptor induction, and homing to small intestine
Integrins...
LPAM-1 (lymphocyte Peyer's patch high endothelial venule adhesion mol. 1); expression by gut-homing T-cells is induced by GALT-derived dendritic cells
Dendritic cell...
lymph node; requirement for GALT-derived dendritic cells in T-cell activation, CCR9 receptor induction, and homing to small intestine
Cell migration...
lymphocyte homing; requirement for GALT-derived dendritic cells in T-cell activation, CCR9 receptor induction, and homing to small intestine
CD8-positive T cell...
requirement for GALT-derived dendritic cells in T-cell activation, CCR9 receptor induction, and homing to small intestine
Intestine...
small, epithelium; requirement for GALT-derived dendritic cells in T-cell activation, CCR9 receptor induction, and homing to small intestine
Cell activation...
T cell; requirement for GALT-derived dendritic cells in T-cell activation, CCR9 receptor induction, and homing to small intestine
Receptors...
TLR-3 (Toll-like receptor-3); homing of T-cells to intestine in relation to GALT-derived dendritic cell maturation in response to
CAS REGISTRY NUMBERS:
24939-03-5 26301-44-0 homing of T-cells to intestine in relation to GALT-derived dendritic cell maturation in response to

2/7/109 (Item 11 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

139021040 CA: 139(2)21040s PATENT
Methods for treating cancer
INVENTOR(AUTHOR): Vicari, Alain P.; Caux, Christophe
LOCATION: USA
ASSIGNEE: Schering Corporation
PATENT: PCT International ; WO 200345431 A2 DATE: 20030605
APPLICATION: WO 2002US38098 (20021126) *US PV333434 (20011127)
PAGES: 47 pp. CODEN: PIXXD2 LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: A61K-039/395A; A61K-047/48B; A61K-031/7088B; A61K-031/405B; A61K-039/00B; A61K-038/19B
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CZ; DE; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; HR; HU; ID; IL; IN; IS; JP; KG; KR; KZ; LC; LK; LR; LT; LU; LV; MA; MD; MG; MK; MN; MX; MZ; NO; NZ; PH; PL; PT; RO; RU; SE; SG; SI; SK; SL; TJ; TM; TN; TR; TT; TZ; UA; UZ; VC; VN; YU; ZA; ZM; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM
DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZM; ZW; AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; SK; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD;

TG
SECTION:
CA215008 Immunochemistry
CA201XXX Pharmacology
CA202XXX Mammalian Hormones
CA214XXX Mammalian Pathological Biochemistry
IDENTIFIERS: cancer immunotherapy dendritic cell TLR receptor
DESCRIPTORS:
Antitumor agents...
agonists of Toll-like receptors and antagonists of tumor-derived inhibitory factors for dendritic cells
Dendritic cell... Mammalia... Human...
agonists of Toll-like receptors and antagonists of tumor-derived inhibitory factors for dendritic cells for cancer immunotherapy
Neoplasm... Melanoma... Mammary gland,neoplasm... Pancreas,neoplasm... Lung,neoplasm... Neuroglia,neoplasm... Stomach,neoplasm... Intestine,neoplasm... Kidney,neoplasm... Prostate gland,neoplasm... Thyroid gland,neoplasm... Ovary,neoplasm... Testis,neoplasm... Head,neoplasm... Esophagus,neoplasm... Eye,neoplasm... Bladder,neoplasm...
agonists of Toll-like receptors and antagonists of tumor-derived inhibitory factors for dendritic cells for treatment of
Interferons...
.alpha.; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Bacteria(Eubacteria)...
as agonists of Toll-like receptor 9
Antibodies... Antisense oligonucleotides...
as antagonists of tumor-derived inhibitory factors for dendritic cells
Transforming growth factors...
.beta.-; immunotherapy of cancer with Toll-like receptor agonists and antagonists of
Chemokines...
CCL16; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Chemokines...
CCL25; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Glycoproteins...
CD40-L (antigen CD40 ligand); in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Intestine,neoplasm...
colon, carcinoma; agonists of Toll-like receptors and antagonists of tumor-derived inhibitory factors for dendritic cells for treatment of
Intestine,neoplasm...
colorectal; agonists of Toll-like receptors and antagonists of tumor-derived inhibitory factors for dendritic cells for treatment of
Oligonucleotides... Phosphorothioate oligonucleotides...
CpG-contg.; as agonists of Toll-like receptor 9 for cancer immunotherapy
Antigens...
DDC; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Uterus,neoplasm...
endometrium; agonists of Toll-like receptors and antagonists of tumor-derived inhibitory factors for dendritic cells for treatment of
Hemopoietins...
FLT3 ligand; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Immunoglobulins...
fragments; as antagonists of tumor-derived inhibitory factors for dendritic cells
Neuroglia,neoplasm...
glioblastoma; agonists of Toll-like receptors and antagonists of tumor-derived inhibitory factors for dendritic cells for treatment of
Liver,neoplasm...
hepatoma; agonists of Toll-like receptors and antagonists of tumor-derived inhibitory factors for dendritic cells for treatment of
Antigens...
HKer 8; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Chemokines...
I-TAC; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Interleukin receptors...
IL-10R; immunotherapy of cancer with Toll-like receptor agonists and antagonists of
Interleukin 6... CTLA-4(antigen)... Gangliosides... Interleukin 10...
immunotherapy of cancer with Toll-like receptor agonists and antagonists of
Carcinoembryonic antigen... Hepatocyte growth factor receptors... Prostate-specific antigen... .alpha.-Fetoproteins... p53(protein)... Tumor necrosis factors... Cytokines... Chemokines... Macrophage inflammatory protein 1.alpha.... RANTES(chemokine)... Monocyte chemoattractant protein-3 ... Monocyte chemoattractant protein-2... Monocyte chemoattractant protein-1... Monocyte chemoattractant protein-4...
in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Chemokines...
interferon .gamma.-inducible protein-10; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Drug delivery systems...
liposomes, cationic; for delivery of immunostimulatory oligonucleotides in cancer immunotherapy
Chemokines...
macrophage inflammatory protein 3.alpha.; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Antigens...
MAGE-1 (melanoma-assocd. antigen 1); in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Antigens...
MAGE-12; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Antigens...
MAGE-2 (melanoma-assocd. antigen 2); in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Antigens...
MAGE-3 (melanoma-assocd. antigen 3); in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Antigens...
MAGE-4; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Antigens...
MART-1; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Cell adhesion molecules...
MCAM (melanoma cell adhesion mol.); in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Transferrins...
melanotransferrins; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Neoplasm...
metastasis; agonists of Toll-like receptors and antagonists of tumor-derived inhibitory factors for dendritic cells for treatment of
Chemokines...
Mig (monokine induced by interferon-.gamma.); in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Antibodies...
monoclonal; as antagonists of tumor-derived inhibitory factors for dendritic cells
Mucins...
MUC1; in combination with TLR9 receptor agonists and antagonists of

tumor-derived inhibitory factors for cancer immunotherapy
Mucins...
MUC2; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Mucins...
MUC3; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Mucins...
MUC4; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Neck,anatomical...
neoplasm; agonists of Toll-like receptors and antagonists of tumor-derived inhibitory factors for dendritic cells for treatment of
Antigens...
NY-ESO-1; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Fusion proteins(chimeric proteins)...
of TLR9 receptor agonists and tumor antigens for cancer immunotherapy
Antigens...
OX-40; immunotherapy of cancer with Toll-like receptor agonists and antagonists of
Antigens...
pMel 17; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Chemokines...
SDF-1 (stromal-derived factor-1); in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Chemokines...
SLC (secondary lymphoid tissue chemokine); in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Drug delivery systems...
slow-release; for delivery of TLR9 receptor agonists and/or antagonists of tumor-derived inhibitory factors
Receptors...
TLR-9 (Toll-like receptor-9); immunotherapy of cancer with antagonists of tumor-derived inhibitory factors for dendritic cells and agonists of
Antigens...
tumor-assocd.; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
Keratins...
19; in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
CAS REGISTRY NUMBERS:
538410-28-5 538410-29-6 538410-30-9 538410-31-0 as agonists of Toll-like receptor 9 for cancer immunotherapy
127464-60-2 363-24-6 81627-83-0 immunotherapy of cancer with Toll-like receptor agonists and antagonists of
9002-10-2 9002-61-3 9074-87-7 9031-28-1 120178-12-3 207621-35-0 83869-56-1 143011-72-7 in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
1811-31-0D tumor antigens-contg., in combination with TLR9 receptor agonists and antagonists of tumor-derived inhibitory factors for cancer immunotherapy
538455-70-8 unclaimed nucleotide sequence; methods for treating cancer
538455-71-9 unclaimed sequence; methods for treating cancer

2/7/110 (Item 12 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

138054529 CA: 138(5)54529r PATENT
Method for monitoring the rate of t-cells recently emigrated from the thymus
INVENTOR(AUTHOR): Olaussen, Richard W.
LOCATION: Norway
ASSIGNEE: Medinnova SF
PATENT: PCT International ; WO 200301209 A1 DATE: 20030103
APPLICATION: WO 2002NO218 (20020619) *NO 20013192 (20010625)
PAGES: 17 pp. CODEN: PIXXD2 LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: G01N-033/569A
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; OM; PH; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TN; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZM; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW ; MZ; SD; SL; SZ; TZ; UG; ZM; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG
SECTION:
CA215001 Immunochemistry
IDENTIFIERS: T lymphocyte thymus CCR9 chemokine receptor
DESCRIPTORS:
Chemokines...
C-C, TECK; method for monitoring the rate of t-cells recently emigrated from the thymus
Chemokine receptors...
CCR9; method for monitoring the rate of t-cells recently emigrated from the thymus
Development,mammalian postnatal...
child; method for monitoring the rate of t-cells recently emigrated from the thymus
Lymphocyte...
homing; method for monitoring the rate of t-cells recently emigrated from the thymus
Cell migration...
lymphocyte homing; method for monitoring the rate of t-cells recently emigrated from the thymus
CD4-positive T cell... Thymus gland... Digestive tract... Aging,animal...
Human... Hematopoietic precursor cell... Transplant and Transplantation...
Human immunodeficiency virus 1... Bone marrow... CD3(antigen)...
CD4(antigen)... CD8(antigen)... CD2(antigen)... CD7(antigen)...
CD45RA(antigen)... Test kits...
method for monitoring the rate of t-cells recently emigrated from the thymus
Intestine...
small, epithelium; method for monitoring the rate of t-cells recently emigrated from the thymus
Intestine...
small, mucosa; method for monitoring the rate of t-cells recently emigrated from the thymus

2/7/111 (Item 13 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

138003690 CA: 138(1)3690d PATENT
Alternative splicing variant of CC chemokine TECK from human thymus, an inhibitor of TECK binding to G protein-coupled receptor GPR-9-6/CC chemokine receptor 9 (CCR9)
INVENTOR(AUTHOR): Yoshie, Osamu; Hiejima, Kunio
LOCATION: Japan,
ASSIGNEE: Teijin Ltd.
PATENT: Japan Kokai Tokkyo Koho ; JP 2002345487 A2 DATE: 20021203
APPLICATION: JP 2001263101 (20010831) *JP 2000268647 (20000905)
PAGES: 9 pp. CODEN: JKXXAF LANGUAGE: Japanese
PATENT CLASSIFICATIONS:
CLASS: C12N-015/09A; A61K-038/00B; A61K-048/00B; A61P-001/00B; A61P-001/04B; A61P-029/00B; A61P-035/00B; A61P-037/06B; A61P-037/08B; A61P-043/00B; C07K-014/47B; C07K-016/18B; C12N-001/15B; C12N-001/19B; C12N-001/21B; C12N-005/10B; G01N-033/15B; G01N-033/50B; G01N-033/53B; G01N-033/566B
SECTION:

CA215005 Immunochemistry
CA203XXX Biochemical Genetics
CA213XXX Mammalian Biochemistry
IDENTIFIERS: CC chemokine TECK splicing variant cDNA sequence human, splicing CC chemokine TECK thymus inhibitor receptor CCR9, G protein coupled receptor 9 CC chemokine TECK binding
DESCRIPTORS:
Antibodies...
against TECK variant; alternative splicing variant of CC chemokine TECK from human thymus, an inhibitor of TECK binding to G protein-coupled receptor GPR-9-6/CC chemokine receptor 9 (CCR9)
Protein sequences... cDNA sequences... Human... Molecular cloning... Drug screening... Molecular association...
alternative splicing variant of CC chemokine TECK from human thymus, an inhibitor of TECK binding to G protein-coupled receptor GPR-9-6/CC chemokine receptor 9 (CCR9)
RNA splicing...
alternative; alternative splicing variant of CC chemokine TECK from human thymus, an inhibitor of TECK binding to G protein-coupled receptor GPR-9-6/CC chemokine receptor 9 (CCR9)
Chemokine receptors... G protein-coupled receptors...
CCR9; alternative splicing variant of CC chemokine TECK from human thymus, an inhibitor of TECK binding to G protein-coupled receptor GPR-9-6/CC chemokine receptor 9 (CCR9)
Chemotaxis...
of leukocytes, TECK-CCR9 mediated, regulation of; alternative splicing variant of CC chemokine TECK from human thymus, an inhibitor of TECK binding to G protein-coupled receptor GPR-9-6/CC chemokine r
Intestine...
small, TECK variant expression in; alternative splicing variant of CC chemokine TECK from human thymus, an inhibitor of TECK binding to G protein-coupled receptor GPR-9-6/CC chemokine receptor 9 (CCR9
Chemokines...
TECK (thymus-expressed chemokine); alternative splicing variant of CC chemokine TECK from human thymus, an inhibitor of TECK binding to G protein-coupled receptor GPR-9-6/CC chemokine receptor 9 (CCR9
Thymus gland...
TECK variant expression in; alternative splicing variant of CC chemokine TECK from human thymus, an inhibitor of TECK binding to G protein-coupled receptor GPR-9-6/CC chemokine receptor 9 (CCR9)
Leukocyte...
TECK-CCR9 mediated chemotaxis of, regulation of; alternative splicing variant of CC chemokine TECK from human thymus, an inhibitor of TECK binding to G protein-coupled receptor GPR-9-6/CC chemokine re
Animal cell line...
293, recombinant expression in; alternative splicing variant of CC chemokine TECK from human thymus, an inhibitor of TECK binding to G protein-coupled receptor GPR-9-6/CC chemokine receptor 9 (CCR9)
CAS REGISTRY NUMBERS:
477266-37-8P amino acid sequence; alternative splicing variant of CC chemokine TECK from human thymus, an inhibitor of TECK binding to G protein-coupled receptor GPR-9-6/CC chemokine receptor 9 (CCR9)
477266-38-9 nucleotide sequence; alternative splicing variant of CC chemokine TECK from human thymus, an inhibitor of TECK binding to G protein-coupled receptor GPR-9-6/CC chemokine receptor 9 (CCR9)
477272-06-3 477272-07-4 477272-08-5 unclaimed nucleotide sequence; alternative splicing variant of CC chemokine TECK from human thymus, an inhibitor of TECK binding to G protein-coupled receptor GPR-9-6/CC chemokine receptor 9 (CCR9)
477272-05-2 unclaimed protein sequence; alternative splicing variant of CC chemokine TECK from human thymus, an inhibitor of TECK binding to G protein-coupled receptor GPR-9-6/CC chemokine receptor 9 (CCR9)

2/7/112 (Item 14 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

136084625 CA: 136(6)84625v JOURNAL
Biological insights into TCR.gamma..delta.+ and TCR.alpha..beta.+ intraepithelial lymphocytes provided by serial analysis of gene expression (SAGE)
AUTHOR(S): Shires, John; Theodoridis, Efstathios; Hayday, Adrian C.
LOCATION: Peter Gorer Department of Immunobiology Guy's, King's, Medical School King's College, University of London, London, UK, SE1 9RT
JOURNAL: Immunity (Immunity) DATE: 2001 VOLUME: 15 NUMBER: 3 PAGES: 419-434 CODEN: IUNIEH ISSN: 1074-7613 LANGUAGE: English PUBLISHER: Cell Press
SECTION:
CA215010 Immunochemistry
IDENTIFIERS: intraepithelial lymphocyte gene expression
DESCRIPTORS:
Proteins...
actin-binding, 1A; serial anal. of gene expression in intraepithelial lymphocytes
Interleukin 2 receptors...
.alpha.-chain; serial anal. of gene expression in intraepithelial lymphocytes
Integrins...
.alpha.E; serial anal. of gene expression in intraepithelial lymphocytes
Transcription factors...
ATF-1; serial anal. of gene expression in intraepithelial lymphocytes
Transcription factors...
ATF-4; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
axins; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
B-cell translocation gene 1; serial anal. of gene expression in intraepithelial lymphocytes
Lymphotoxin...
.beta.; serial anal. of gene expression in intraepithelial lymphocytes
Actins... Transforming growth factors...
.beta.-; serial anal. of gene expression in intraepithelial lymphocytes
Interleukin 2 receptors...
.beta.-chain; serial anal. of gene expression in intraepithelial lymphocytes
G proteins(guanine nucleotide-binding proteins)...
.beta.-2; serial anal. of gene expression in intraepithelial lymphocytes
Transforming growth factors...
.beta.1-; serial anal. of gene expression in intraepithelial lymphocytes
Microglobulins... Transforming growth factors...
.beta.2-; serial anal. of gene expression in intraepithelial lymphocytes
Integrins...
.beta.3; serial anal. of gene expression in intraepithelial lymphocytes
Transforming growth factors...
.beta.3-; serial anal. of gene expression in intraepithelial lymphocytes
Transforming growth factors...
.beta.4-; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
BY56; serial anal. of gene expression in intraepithelial lymphocytes
Chemokines...
C-C, LARC (liver and activation regulated chemokine); serial anal. of gene expression in intraepithelial lymphocytes
Chemokines...
C-C, TARC (thymus and activation-regulated chemokine); serial anal. of gene expression in intraepithelial lymphocytes
Chemokines...
C-C, TECK; serial anal. of gene expression in intraepithelial lymphocytes
Transcription factors...
c-fos; serial anal. of gene expression in intraepithelial lymphocytes
Transcription factors...
c-jun; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...

calgizzarins; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
calpactins; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
calponins, 2; serial anal. of gene expression in intraepithelial lymphocytes
Transcription factors...
CBP interacting transactivator 4; serial anal. of gene expression in intraepithelial lymphocytes
Antigens...
CD48; serial anal. of gene expression in intraepithelial lymphocytes
CD antigens...
CD63; serial anal. of gene expression in intraepithelial lymphocytes
CD antigens...
CD94; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
cellular apoptosis susceptibility protein; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
Chat H SH2-contg. protein 3; serial anal. of gene expression in intraepithelial lymphocytes
Transcription factors...
chromobox homolog 4; serial anal. of gene expression in intraepithelial lymphocytes
Neurotrophic factor receptors...
ciliary; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
DAP10; serial anal. of gene expression in intraepithelial lymphocytes
Phosphoproteins...
DAP12 (DNAX activation protein 12); serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
Deltex; serial anal. of gene expression in intraepithelial lymphocytes
Elongation factors(protein formation)...
eEF-1.alpha.; serial anal. of gene expression in intraepithelial lymphocytes
Elongation factors(protein formation)...
eEF-2; serial anal. of gene expression in intraepithelial lymphocytes
Transcription factors...
ELF-1; serial anal. of gene expression in intraepithelial lymphocytes
Transcription factors...
embigin; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
FGF-inducible 14; serial anal. of gene expression in intraepithelial lymphocytes
Hemopoietins...
FLT3 ligand; serial anal. of gene expression in intraepithelial lymphocytes
Chemokines...
fractalkines; serial anal. of gene expression in intraepithelial lymphocytes
Interferons...
.gamma.; serial anal. of gene expression in intraepithelial lymphocytes
Actins...
.gamma.-actins; serial anal. of gene expression in intraepithelial lymphocytes
Interleukin 2 receptors...
.gamma.-chain; serial anal. of gene expression in intraepithelial lymphocytes
Interferon receptors...
.gamma.-interferon; serial anal. of gene expression in intraepithelial lymphocytes
Transcription factors...
GATA-3; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
GDP-dissocn. inhibitor; serial anal. of gene expression in intraepithelial lymphocytes
G proteins(guanine nucleotide-binding proteins)...
gene CDC42; serial anal. of gene expression in intraepithelial lymphocytes
G proteins(guanine nucleotide-binding proteins)...
gene rac2; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
Grb-2; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
Grb10; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
guanine nucleotide-binding protein .alpha. stimulating; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
guanine nucleotide-binding protein .beta.2-related sequence 1; serial anal. of gene expression in intraepithelial lymphocytes
Histocompatibility antigens...
H-2, class I; serial anal. of gene expression in intraepithelial lymphocytes
Growth factors,animal...
hepatoma-derived growth factors; serial anal. of gene expression in intraepithelial lymphocytes
High-mobility group proteins...
HMG2; serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
HS1; serial anal. of gene expression in intraepithelial lymphocytes
Receptors...
ICOS (inducible co-stimulator); serial anal. of gene expression in intraepithelial lymphocytes
Proteins...
ICOS ligand; serial anal. of gene expression in intraepithelial lymphocytes
Integrins... CD antigens...
integrin .beta.7; serial anal. of gene expression in intraepithelial lymphocytes
Interleukin receptors...
interleukin 10 receptors; serial anal. of gene expression in intraepithelial lymphocytes
Interleukin receptors...
interleukin 12; serial anal. of gene expression in intraepithelial lymphocytes
CAS REGISTRY NUMBERS:
362479-32-1 .alpha. isoform; serial anal. of gene expression in intraepithelial lymphocytes
9001-16-5 C; serial anal. of gene expression in intraepithelial lymphocytes
95076-93-0 isoform A; serial anal. of gene expression in intraepithelial lymphocytes
78990-62-2 isoform 2; serial anal. of gene expression in intraepithelial lymphocytes
143180-73-8 387877-30-7 143180-74-9 9001-51-8 9001-50-7 198154-07-3 144377-03-7 143550-91-8 156476-39-0 143749-56-8 67763-96-6 148348-15-6 114051-78-4 300859-91-0 148640-14-6 115926-52-8 138674-26-7 157482-36-5 153190-61-5 152478-57-4 152478-56-3 386278-22-4 9001-58-5 101149-94-4 148047-29-4 195329-69-2 148047-34-1 serial anal. of gene expression in intraepithelial lymphocytes

2/7/113 (Item 15 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

133348920 CA: 133(25)348920x JOURNAL
Cutting edge: A novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in mucosal tissues
AUTHOR(S): Pan, Junliang; Kunkel, Eric J.; Gosslar, Uwe; Lazarus, Nicole; Langdon, Patricia; Broadwell, Kim; Vierra, Mark A.; Genovese, Mark C.; Butcher, Eugene C.; Soler, Dulce
LOCATION: Laboratory of Immunology and Vascular Biology, Department of Pathology, Stanford University School of Medicine, Stanford, CA, 94305, USA

JOURNAL: J. Immunol. DATE: 2000 VOLUME: 165 NUMBER: 6 PAGES: 2943-2949 CODEN: JOIMA3 ISSN: 0022-1767 LANGUAGE: English PUBLISHER: American Association of Immunologists
SECTION:
CA215005 Immunochemistry
CA203XXX Biochemical Genetics
IDENTIFIERS: chemokine MEC cDNA sequence human activity, mucosae assocd epithelial chemokine cDNA sequence human activity, CCR10 CCR3 receptor ligand chemokine MEC
DESCRIPTORS:
Chemokine receptors...
.beta., CCR10 (cysteine-cysteine chemokine receptor 10); cDNA sequence of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues
Chemokine receptors...
C-C CKR-3 (cysteine-cysteine chemokine receptor 3); cDNA sequence of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues
cDNA sequences... Protein sequences...
cDNA sequence of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues
Intestine...
colon, epithelium; tissue-specific mRNA expression of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues
Chemokines...
CTACK; protein sequence homol. of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues with chemokines CTACK and TECK
Lung... Lymph node... Mammary gland... Placenta... Salivary gland... Skin ... Stomach... Trachea(anatomical)...
epithelium; tissue-specific mRNA expression of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues
Embryo,animal...
fetus; tissue-specific mRNA expression of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues
Protein sequences...
homol.; protein sequence homol. of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues with chemokines CTACK and TECK
Intestine...
ileum, epithelium; tissue-specific mRNA expression of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues
Intestine...
jejunum, epithelium; tissue-specific mRNA expression of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues
CD4-positive T cell... Chemotaxis... Eosinophil... Lymphocyte...
lymphoid cell chemotaxis response to novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues
Chemokines...
MEC (mucosae-assocd. epithelial chemokine); cDNA sequence of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues
T cell(lymphocyte)...
memory; lymphoid cell chemotaxis response to novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues
Intestine...
rectum, epithelium; tissue-specific mRNA expression of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues
Intestine...
small, epithelium; tissue-specific mRNA expression of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues
Chemokines...
TECK; protein sequence homol. of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues with chemokines CTACK and TECK
Gene,animal... mRNA... Transcription,genetic...
tissue-specific mRNA expression of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues
CAS REGISTRY NUMBERS:
208733-73-7 amino acid sequence; cDNA sequence of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues
291743-00-5 nucleotide sequence; cDNA sequence of novel chemokine ligand for CCR10 and CCR3 expressed by epithelial cells in human mucosal tissues

2/7/114 (Item 16 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

133251279 CA: 133(18)251279g PATENT
Anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
INVENTOR(AUTHOR): Andrew, David P.; Zabel, Brian A.; Ponath, Paul D.
LOCATION: USA
ASSIGNEE: Leukosite, Inc.
PATENT: PCT International ; WO 200053635 A1 DATE: 20000914
APPLICATION: WO 2000US6240 (20000310) *US 266464 (19990311)
PAGES: 125 pp. CODEN: PIXXD2 LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: C07K-016/24A; C07K-016/28B; C12N-005/10B; C12N-005/20B; G01N-033/53B; G01N-033/577B; A61K-039/395B; A61P-037/00B; A61P-035/00B
DESIGNATED COUNTRIES: AE; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CR; CU; CZ; DE; DK; DM; DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM
DESIGNATED REGIONAL: GH; GM; KE; LS; MW; SD; SL; SZ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG
SECTION:
CA215005 Immunochemistry
CA201XXX Pharmacology
CA209XXX Biochemical Methods
CA263XXX Pharmaceuticals
IDENTIFIERS: antibody CC chemokine receptor GPR96 TECK, inflammation cancer antibody fragment GPR96 TECK
DESCRIPTORS:
Animal cell line... Animal cell... Antibodies... Chemiluminescent substances... Chemotaxis... Cytotoxic agents... DNA sequences... Drug screening... Drugs... Enzymes,biological studies... Epitopes... Fluorescent substances... Fusion proteins(chimeric proteins)... G protein-coupled receptors... Immunoglobulins... Inflammation... Labels... Ligands...
Neoplasm... Protein sequences... Radionuclides...
anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
Samples...
biol.; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
Chemokine receptors...
C-C (cysteine-cysteine chemokine receptors), GPR-9-6/CCR9; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
Intestine,disease...
colitis; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
Intestine,disease...
Crohn's; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
Immunoglobulins...
fragments; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
Leukocyte...

homing; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
B cell(lymphocyte)...
immortalized; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
Drug delivery systems...
immunoconjugates; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
Test kits...
immunodiagnostic; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
Intestine,disease...
inflammatory; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
Transport properties...
ionic, Ca+2; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
Animal cell line...
Molt 4; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
Animal cell line...
MOLT13; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
Rodent...
murine; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
Hybridoma...
3C3; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
CAS REGISTRY NUMBERS:
197028-91-4 201169-36-0 264863-85-6 294216-09-4 294216-10-7 amino acid sequence; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
7440-70-2 biological studies, flux; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
175111-11-2 294216-07-2 294216-08-3 nucleotide sequence; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
294216-12-9 294216-13-0 294216-14-1 294216-15-2 294216-16-3 294216-17-4 294216-18-5 294216-19-6 unclaimed nucleotide sequence; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions
294211-61-3 unclaimed sequence; anti-GPR-9-6 and anti-TECK antibodies and methods of identifying modulators of GPR-9-6 and TECK functions

2/7/115 (Item 17 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

132106969 CA: 132(9)106969g PATENT
Chemokines as adjuvants of immune response
INVENTOR(AUTHOR): Caux, Christophe; Vanbervliet, Beatrice; Lebecque, Serge; Vicari, Alain; Dieu, Marie-Caroline
LOCATION: Fr.
ASSIGNEE: Schering-Plough
PATENT: European Pat. Appl. ; EP 974357 A1 DATE: 20000126
APPLICATION: EP 98401799 (19980716)
PAGES: 16 pp. CODEN: EPXXDW LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: A61K-038/19A
DESIGNATED COUNTRIES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IT; LI; LU; NL; SE; MC; PT; IE; SI; LT; LV; FI; RO
SECTION:
CA215005 Immunochemistry
CA203XXX Biochemical Genetics
CA263XXX Pharmaceuticals
IDENTIFIERS: chemokine cytokine immune adjuvant antigen vaccine, cancer autoimmune disease infection graft rejection
DESCRIPTORS:
Immunostimulants...
adjuvants; chemokines as adjuvants for inducing antigen-specific immune response
Antibodies...
anti-CD40; chemokines as adjuvants for inducing antigen-specific immune response
Animal virus... Bacteria(Eubacteria)... Fungi...
antigen; chemokines as adjuvants for inducing antigen-specific immune response
Immunity...
antigen-specific; chemokines as adjuvants for inducing antigen-specific immune response
Infection...
bacterial; chemokines as adjuvants for inducing antigen-specific immune response
Allergy... Antigen presentation... Antigens... Autoimmune disease... Carcinoembryonic antigen... CD40(antigen)... Cell migration... Chemokines ... Cytokines... Dendritic cell... Eye,neoplasm... Genetic vectors... Hepatocyte growth factor receptors... Interleukin 4... Intestine,neoplasm ... Kidney,neoplasm... Liver,neoplasm... Lung,neoplasm... Macrophage inflammatory protein 1.alpha.... Macrophage inflammatory protein 1.beta.... Melanoma... Neoplasm... Ovary,neoplasm... Pancreas,neoplasm... Prostate-specific antigen... RANTES(chemokine)... Stomach,neoplasm... Testis,neoplasm... Thyroid gland,neoplasm... Transplant rejection... Tumor necrosis factors... .alpha.-Fetoproteins...
chemokines as adjuvants for inducing antigen-specific immune response
Intestine,neoplasm...
colon; chemokines as adjuvants for inducing antigen-specific immune response
Intestine,neoplasm...
colorectal; chemokines as adjuvants for inducing antigen-specific immune response
Nucleic acids...
CpG motif-contg.; chemokines as adjuvants for inducing antigen-specific immune response
Chemokines...
DC tactin .beta.; chemokines as adjuvants for inducing antigen-specific immune response
Uterus,neoplasm...
endometrium; chemokines as adjuvants for inducing antigen-specific immune response
Mucins...
episialins; chemokines as adjuvants for inducing antigen-specific immune response
Neuroglia...
glioblastoma; chemokines as adjuvants for inducing antigen-specific immune response
Neuroglia...
glioma; chemokines as adjuvants for inducing antigen-specific immune response
Glycoproteins,specific or class...
gp100; chemokines as adjuvants for inducing antigen-specific immune response
Sialoglycoproteins...
gp75; chemokines as adjuvants for inducing antigen-specific immune response
Liver,neoplasm...
hepatoma; chemokines as adjuvants for inducing antigen-specific immune response
Parasite...
infection; chemokines as adjuvants for inducing antigen-specific immune response
Drug delivery systems...
injections, i.m.; chemokines as adjuvants for inducing antigen-specific immune response
Drug delivery systems...
injections, s.c.; chemokines as adjuvants for inducing antigen-specific immune response
Drug delivery systems...
intradermal; chemokines as adjuvants for inducing antigen-specific

immune response
Organ,animal...
lymphoid; chemokines as adjuvants for inducing antigen-specific immune response
Cytokines...
macrophage inflammatory protein, 3.alpha.; chemokines as adjuvants for inducing antigen-specific immune response
Chemokines...
MDC; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
melanoma-assocd.; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
melanoma-assocd., high mol. wt.; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
melanoma-assocd., melan A; chemokines as adjuvants for inducing antigen-specific immune response
Transferrins...
melanotransferrins; chemokines as adjuvants for inducing antigen-specific immune response
Carcinoma...
metastatic; chemokines as adjuvants for inducing antigen-specific immune response
Chemokines...
monocyte chemoattractant protein 3; chemokines as adjuvants for inducing antigen-specific immune response
Cytokines...
monocyte chemoattractant protein 4; chemokines as adjuvants for inducing antigen-specific immune response
Mucins...
MUC 2; chemokines as adjuvants for inducing antigen-specific immune response
Mucins...
MUC 3; chemokines as adjuvants for inducing antigen-specific immune response
Mucins...
MUC 4; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
MUC18; chemokines as adjuvants for inducing antigen-specific immune response
Bladder... Esophagus... Head... Mammary gland... Neck,anatomical...
Prostate gland...
neoplasm; chemokines as adjuvants for inducing antigen-specific immune response
Cytokines...
RP-105; chemokines as adjuvants for inducing antigen-specific immune response
Chemokines...
SDF-1; chemokines as adjuvants for inducing antigen-specific immune response
Chemokines...
Teck; chemokines as adjuvants for inducing antigen-specific immune response
Drug delivery systems...
topical; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
tumor-assocd.; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
tumor-assocd., DDC; chemiokines as adjuvants for inducing antigen-specific immune response
Antigens...
tumor-assocd., Hker 8; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
tumor-assocd., k19; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
tumor-assocd., MAGE-1; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
tumor-assocd., MAGE-12; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
tumor-assocd., MAGE-2; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
tumor-assocd., MAGE-3; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
tumor-assocd., MAGE-4; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
tumor-assocd., MART-1; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
tumor-assocd., pMel 17; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
tumor-assocd., prostate specific membrane antigen; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
tumor-assocd., Tyr1; chemokines as adjuvants for inducing antigen-specific immune response
Antigens...
tumor-assocd., Tyr2; chemokines as adjuvants for inducing antigen-specific immune response
Infection...
viral; chemokines as adjuvants for inducing antigen-specific immune response
CAS REGISTRY NUMBERS:
9002-10-2 9002-61-3 9031-28-1 14215-68-0 83869-56-1 chemokines as adjuvants for inducing antigen-specific immune response
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$1.61 0.475 DialUnits File155
$9.68 44 Type(s) in Format 7
$9.68 44 Types
$11.29 Estimated cost File155
$3.41 0.568 DialUnits File5
$68.20 31 Type(s) in Format 7
$68.20 31 Types
$71.61 Estimated cost File5
$2.32 0.250 DialUnits File71
$22.50 10 Type(s) in Format 7
$22.50 10 Types
$24.82 Estimated cost File71
$5.13 0.431 DialUnits File73
$19.80 6 Type(s) in Format 7
$19.80 6 Types
$24.93 Estimated cost File73
$12.35 0.537 DialUnits File357
$28.70 7 Type(s) in Format 7
$28.70 7 Types
$41.05 Estimated cost File357
$7.21 0.575 DialUnits File399
$46.75 17 Type(s) in Format 7
$46.75 17 Types
$53.96 Estimated cost File399
OneSearch, 6 files, 2.835 DialUnits FileOS
$0.53 TELNET
$228.19 Estimated cost this search
$232.18 Estimated total session cost 4.096 DialUnits
Logoff: level 05.15.00 D 09:04:29